WAYNE O. KESTER
Brigadier General, USAF, (VC)Ret.
Route 3, 14 Hillcrest Circle
Golden, Colorado

Proceedings

of the

Seventh Annual American Association of Equine Practitioners Convention

FORT WORTH, TEXAS

December, 1961

XUM

Proceedings

of the

Seventh Annual

American Association

of

Equine Practitioners

Convention

HOTEL TEXAS,
FORT WORTH, TEXAS

December 3 thru 5, 1961

Permission to reprint material in this book
may be obtained under certain conditions.

XUM

FOREWORD

This is the sixth annual proceedings book of the American Association of Equine Practitioners covering the seventh annual convention held in Fort Worth, Texas, December 3 through 5, 1961.

The proceedings of the business sessions were transcribed and are a separate matter of record. For purposes of brevity and economy, this book is confined to an edited version of the scientific papers, panels, open discussions and the banquet address.

Since the address by Colonel Fred Maurer on African Horsesickness was largely an extemporaneously delivered commentary on color slides, it was considered impracticable to incorporate it in this book, however, an abstract of Colonel Maurer's remarks will be found in the appropriate section.

The final contribution on the program was an illustrated travelogue, with an equine bias, by Dr. T. E. Dunkin. At the time of going to press, no abstract of this talk was available.

Dr. Mark L. Morris, President of the American Veterinary Medical Association, delivered the major address at the annual banquet.

F. J. MILNE, D.V.M.
Editor.

XUM

DR. M. B. TEIGLAND, President
American Association of Equine Practitioners,
1961.

1961
OFFICERS

Chairman of the Board of Directors

BRIGADIER-GENERAL WAYNE O. KESTER, USAF (Retd.)

Denver, Colorado

President

DR. M. B. TEIGLAND,

Opa Locka, Florida

President-Elect

DR. W. O. REED,

Elmont, New York

Secretary-Treasurer

DR. F. J. MILNE,

Guelph, Ontario, Canada

Executive Secretary

DR. MARION L. SCOTT,

Akron, Ohio

XUM

BANQUET ADDRESS

by

MARK L. MORRIS, President, A.V.M.A.

The horse is here to stay. This animal, closely associated with man since prehistoric times will never cease to exist. For years the public has been waiting for the day when the few remaining horses would be relegated to zoological gardens; there put on exhibit as a reminder of the contribution made in the development of civilization. With an exploding population comes an avalanche of interest of the youth of our country in the horse. In 4-H Club projects this year, horse projects are reported to have led all others in interest across the nation. It is evident that youth is expressing an increasingly greater interest in the horse, than in any other animal. These are the leaders of tomorrow. Their interests therefore are important. There is a tremendous expansion of all pleasure industries and the horse is an important segment of such activities. More people are living in urban areas and seek opportunities for outdoor recreation which provides an opportunity to live and work with living things. You, who are equine practitioners, stand at the threshold of a new era in veterinary medicine. The challenges are great. How can this rapidly expanding equine industry best be served? How can the most favorable image of the veterinary profession which you represent be projected? First, we should examine the problem. Step one is to become better prepared to cope with the rapidly changing and expanding field of equine medicine. Here is where careful planning needs to be done. To permit this segment of the profession to grow indiscriminately would be an error and could lead only to discouraging results. Indiscriminate growth will be detrimental not only to the profession but to the horse-owning public as well. It would seem expedient to methodically examine our present position and determine if a concrete program, a "footprint", could be designed for the future. In the public eye, what is the image of the equine practitioner? A prediction could be made; with many people thinking that the horse is disappearing, then the profession servicing the medical needs of this animal should likewise be disappearing. What career counselor or director of secondary education would encourage a bright young student to enter the field of equine medicine? In my opinion, this person would be extremely rare. The average person is not aware that at present there are well educated, competent veterinarians servicing equine medicine and never were their services more needed. Some

XUM

of America's best qualified veterinarians are represented here in this audience. A past president of the A.V.M.A. is Chairman of your Board of Directors; the current president-elect of the American Veterinary Medical Association has been a member of your Board of Directors since the founding of this organization. How can a true image, as we know it, be projected to the horse industry of this country and to the thousands of young men and women who are currently interested and will continue to be in the future? How can the practice of equine medicine be expanded and improved to serve more efficiently this old but rapidly expanding industry?

ORGANIZATION

A dynamic and effective organization employing the services of well-qualified personnel is mandatory, otherwise little can be accomplished in the complex society in which we live today. The equine practitioners through the Equine Practitioners' Association must have a strong and effective organization. In itself it cannot accomplish the objectives. It must be closely linked to veterinary medicine as a whole through the American Veterinary Medical Association and exert its influence at the local level through the state and local organizations. If the Equine Practitioners' Association is not so equipped at the present time, then this, in my opinion, is step one.

PROFESSIONAL PROFICIENCY

The man who owns valuable horses expects sound, expert professional advice. He expects his veterinarian to be thoroughly conversant with the latest diagnostic and therapeutic methods and these include husbandry, management, and nutrition. Effective management of the clinical case will not suffice. This suggests the need for well-organized programs of continuing education. You must decide how these can best be organized and implemented and through what organization such programs can be most effective. There appear to be at least four possibilities. (1) One would be the establishment of a specialty organization which would include a specialty board. This may prove difficult because specialty boards are more often concerned with disciplines rather than with a species. For example, a veterinary pathologist certified by that board could serve perfectly well in the equine area.

(2) It may be possible to further expand and improve the annual meeting to provide further information in the field of equine medicine.

(3) Group seminars organized at the local level can be very effective. This method has worked out well in small animal

medicine. These seminars are developing and spreading, and must be encouraged. Groups of equine practitioners located in certain areas could very well sponsor such programs.

(4) Specialized courses in equine medicine could be encouraged and further developed at the various educational institutions. These could be for a few days or could extend over several weeks. These programs must be tailored to fit the demands of you equine practitioners, and further specialized to the requirements imposed by the needs of various areas.

These four suggestions could be initiated promptly but they require your individual and collective motivation.

CLIENT RELATIONS

In all areas of medical practice, client relations are important. One problem of the physician is that intrinsic relations with his patients are not what they were twenty years ago. When I was a boy, our family had great confidence in our doctor and he was a close friend of the family. Many changes have taken place in our economy and with the application of newer methods in the practice of medicine, this close relationship between the doctor and his patient has changed considerably. This is a serious problem facing the medical profession today. Are we in the veterinary profession letting this happen to us today? Most animal owners like to develop a feeling of closeness and confidence with the veterinarian who is responsible for the health of their animals: this is especially true of companion animals. I had a letter recently from a blind man, a graduate of Yale University, Phi Beta Kappa, and a successful attorney. He has a Seeing-Eye dog for a pair of eyes. He stated, "The period covered by my Seeing-Eye dogs has brought some vast changes in the more practical aspects of veterinarians' service. In the 1930's they had small offices, and all dealings with them were always direct, personal contact. And they were in town where you could walk to the office. Now, all is **BIG BUSINESS** with these so-called animal hospitals out in the country somewhere. They always want you to leave your dog there, come back later, after they have told you by telephone that she has been taken care of. Then, when you get your friend to drive you out there to get your dog, you see some clerical person who takes your name, and after a while, a kennel boy brings your dog; the doctor is busy. And, he is. There is usually a whole flock of cars there, indicating the vast change from the days when most pets recovered from their illnesses naturally, or they died, and that's all there was to it.

"It has been my experience that the veterinarian who has any real idea what our Seeing-Eye dogs mean to us is the exception rather than the rule. This was different in the early days

XUM

when we often went to the veterinarian's office under our own power, but now we usually have to go with a personal guide because of the transportation problem, so they are much less aware of what goes on. The situation becomes just like that of anyone bringing their pet for medical attention.

"There is one thing, however, that's for sure; the veterinarians **now** have had **good** formal training; **no more** of the **home-grown** variety as we had twenty-five years ago."

Isn't this blind man depicting for us a parallel to that which has taken place in the medical profession? Isn't he saying that the veterinarian of today is losing personal contact with his client? Actually, isn't the image of our profession as portrayed by this blind man a factual one? Where do you, the equine practitioner, stand? Do you have good, close liaison and effective personal relationships including good communications with your clients? I refer to the owner of the animal for which you are rendering the service, or do you find it necessary to deal by remote control or a circuitous route through the groom, trainer, stable attendant, or other employee? Are you losing the very thing that the blind man points out that is so important . . . a closer, more personal relationship with your clients? Your proficiency, no matter how perfect, can be no more effective or better than your client relationship.

In my practice where several veterinarians and a number of laymen were employed, we always insisted that if at all possible, a veterinarian receive the patient, speak with the owner personally, at least once, while the animal was hospitalized, and certainly be present when the animal was discharged from the hospital. Laymen performed many functions such as the collection of laboratory samples, feeding, cleaning, husbandry, and nursing, but the veterinarian always dealt personally with the owners. There can be no area in veterinary medicine where this is more important than in equine practice, since not only is there sentiment involved but often large sums of money. It is imperative that the veterinarian communicate directly with the owner on a personal basis especially at the termination of the case. If this is not possible, then the information finally given the lay personnel who are responsible for the animal should be transmitted in the form of a written memorandum directed to the owner with an information copy to attendants in charge. Such technique will not replace personal, face-to-face contact but will assist materially in maintaining good veterinary-owner relations. Do you take time to carefully explain to the owner the intricacies and complexities of the case or in the haste of practice, do you indicate that you will give the horse "a shot" and that he, the owner should let you know if further service is needed? It should be remembered that **stable hands** can give

"shots". You should regulate your professional life to take time to get the owner to understand the problem and what you, a Doctor of Veterinary Medicine, are doing about if. If you fail to explain why you are performing a professional service, then you are neglecting an opportunity to develop a closer, more personal client relationship and perhaps the owner will feel he doesn't really need you to care for his animal.

COMMUNICATIONS

What we have been talking about with respect to client relations is improved and more effective **communications.** Communications is one of our major problems. There are three distinct groups with whom the veterinarian must communicate.

First, the clients, the owners of the horses; second, the trainers, farm managers, stablemen and attendants; and, third, the general public.

Communications with all these groups becomes a major project, thus a discussion of the fundamentals of communication seems in order.

At the recent Conference of State Veterinary Association Secretaries, Professor Kumata, head of the Department of Communications at Michigan State University was one of the speakers. He indicated that knowing when to use the personal and impersonal method is the key to any communications effort. He said there are five stages of action in successful communication: Awareness, Interest, Evaluation, Trial and Adoption. In stimulating awareness and interest, impersonal sources such as publications are effective. To obtain evaluation, trial, and adoption with eventual participation, personal sources are needed. If you wish to encourage the owners of valuable horses to adopt your recommendations and employ your professional services, impersonal techniques **will not be** effective and owner participation can only be accomplished through personal face-to-face contact. It was pointed out that it is a fallacy to assume that all you have to do is inject information to get results. It is essentially impossible to convert people by the impersonal approach; you can push but you cannot change attitudes. Furthermore, most people do that which is easiest. The will read material only if it is readable. You may use the impersonal approach if the audience or the person trusts it, which is not often true. It is obvious, therefore, that if you hope to maintain good liaison with the owners of valuable horses, it can be done **only** through personal contacts. This is especially true if you wish to gain their participation in management programs which you wish adopted. If it is your intent to influence the thinking of the horse-owning public and gain their acceptance of you as an equine practitioner, then this can only be done on

XUM

the personal contact basis. Publications will only serve to create an awareness and an interest. You, the Doctor of Veterinary Medicine, must accomplish the end objective through your personal contact with the owner. This places the responsibility squarely on your shoulders.

PRACTICE TRENDS

In all phases of veterinary medicine, changes are taking place rapidly. There seems to be less need for the routine clinician and more need for the specialist. The consultant and most assuredly, the good diagnostician are needed. We are certainly living in the "do-it-yourself era" with more medicated feeds and vastly more accurate advice and information dispensed by journals, radio, television, and other media. The equine practitioner must recognize these trends and adapt his practice methods to them. The animal owners should become a part of this way of life and not be apart from these trends. Many clients can be trained to effectively take over more emergency and routine work which will benefit everyone involved. For example, in small animal practice, veterinarians are doing less vermifuging, tail docking, ear trimming, and other minor operations and treatments. Some of this is due to a better informed clientele, some to rising costs and fees. I can see the same trend developing in equine medicine as well, and it will work to your ultimate benefit. As this trend develops, we will be able to guide it so that the animal will still have the benefit of all the veterinary attention it needs even though we do not do every routine chore. Therefore, we should give considered attention to teaching our clients and improve first-aid and preventive medicine measures with the thought of making them a **part** of our team rather than **apart** from it.

There is nation-wide interest in expanded partnerships, cooperative and corporate-type practices. This is an area that should command your very careful and thoughtful attention. In a number of states, laws have been or are scheduled to be passed which will permit professional men to function through a corporate structure. This trend embodies certain dangers, for example, a large industrial pharmaceutical firm is establishing free diagnostic laboratories in four states; although at the moment the poultry industry is chiefly involved, this procedure could spread to other species, in fact, it is scheduled to do so in one state. Free service just doesn't exist . . . somebody pays! Free diagnostic laboratories may not be of direct concern to you as an equine practitioner, but it does certainly concern you as a veterinarian. It certainly concerns the conditions under which you are licensed to practice veterinary medicine since it very conceivably can lead to the weakening or the challenging of the legality of the laws under which you are

now practicing your profession. Your state association must keep well-informed on developments.

With the rapid expansion that is taking place in the field of equine medicine, the more efficient orientation of the recent graduate becomes important. The better equine practitioner this man becomes and the more active he is in the organization, the better and more effective your association will be. There is considerable room for improved orientation. It is suggested that the practice now carried on in one state of assigning each young veterinarian to an older practitioner to help guide him until he becomes established, be further expanded. This permits a young man to become familiar and accustomed to the methods and customs peculiar to the state or to the practice and thus to perfect his proficiency. It would seem that this practice of helping to direct and guide these young men would be very beneficial, especially where there are a number of practitioners concentrated in a local area such as at the racetrack, or in areas where there are numerous breeding farms. This is good preventive medicine because a young practitioner who forms good practice habits early, under the guidance of an experienced practitioner, is almost certain to become a credit to the profession.

THE FUTURE OF EQUINE MEDICINE

Like any other great industry, the future of the equine industry and that of equine medicine is directly related to the investment that is made in fundamental research. Every large, successful industry in this country today is underpinned by substantial research and development programs. As I view the equine industry, it seems to me that it has been and is still waiting for someone to step up and make the necessary investments, and to carry the responsibility for a good research program. In an industry as large as the equine industry, this deficiency is difficult to understand. The equine practitioner today is seriously handicapped due to a lack of reliable research data flowing from programs that should have been instituted at least two decades ago. This creates a serious vacuum. At the present time, the chemical physiology of the equine is very poorly understood. A simple example will illustrate. The normal electrolyte balance of the young growing colt has not been well established. When it becomes necessary to re-establish the electrolyte balance through the administration of fluids, this must be done on a clinical basis because the actual requirements are as yet unknown.

In the field of nutrition, great strides have been made employing laboratory animals, thus a great deal is known about the nutrition of the rat, the mouse, and the guinea pig. Considerable information is available about the nutrition of the dog. Little is

XUM

known concerning the basal requirements of the cat. Very little is known concerning the true nutritive requirements of the horse, either for maintenance, gestation, lactation or growth. It is therefore understandable that the veterinary practitioner is often criticized by the horse-owner for being unable to render services which actually have not yet been perfected. The responsibility for this lack of progress, it seems to me, rest squarely at the door of the industry which depends on the services of the veterinary profession. In the cattle industry tremendous sums have been invested at our experiment stations and in private laboratories to make available to the veterinary student, information concerning nutrition and the diseases of livestock. The same is **not** the case with the horse. Thus, how can the young veterinary student be expected to become more knowledgeable in an area where information is non-existent?

It is the responsibility of the veterinary profession to delineate for the equine industry the seriousness of the problem that exists and the tremendous need for research and procedures that can be followed to get the job done. We, the veterinary profession, need to lay out the program and provide the organizational blueprint so that the industry can step in and help create funds for, and the implementation of such an undertaking.

In other words, as I see it, four things are needed. First, an administrative organization that provides a common meeting ground for owners of all breeds and all interests in the equine industry. Second, a clear delineation of the problem and a program outlined for approaching it. The third need is for the funds to support such a continuing research program; and fourth, the administrative supervisory body to follow through and ensure that good research is done and to report the results obtained.

It would seem in examining the over-all picture in retrospect that it is up to this organization, the American Association of Equine Practitioners, to take the lead in plotting the course and provide the motivation to get this great equine industry moving at the rate of progress and in the direction it deserves to go.

XUM

CONTENTS

Foreword i

Officers, 1961 iii

Banquet Address, Mark L. Morris iv

"Quarter Horse Practice"
C. T. Mason 21

"The Surgical Handling of Tissues"
C. Snyder 31

"Common Injuries of the Eye"
W. G. Magrane 43

"Snakebite Poisoning in Horses"
C. H. Burger 71

Clinico-Pathologic Conference 77
Moderator: C. W. Raker
Panel: S. P. Dey
E. Dillman
F. J. Milne

"Follicle Examination of Ovaries in Relation to Optimum Breeding Time"
J. D. Gadd 89

"Periodic Ophthalmia"
W. G. Magrane 99

"Physical Examination of the Horse for Soundness"
J. E. Peters 109

"Nutritional Diseases of Dogs and Their Possible Relationship to the Horse"
W. O. Kester 115

"Some Drugs and Therapy Affecting the Gastro-Intestinal Tract"
S. F. Scheidy and J. E. Martin 121

"African Horsesickness"
F. D. Maurer 131

"Poisoning in the Horse with Specific Reference to Poisonous Plants"
M. E. Fowler 133

"General Principles in Radiology and Interpretation. I.
W. D. Carlson 141

"Recent Advances in Equine Anesthesia"
E. W. Jones 153

Panel: "Foal Diseases" 165
Chairman: W. O. Reed

"Umbilical Hernia and Repair of the Ruptured Bladder"
W. R. McGee

"Infectious Diseases of the Foal"
J. S. M. Cosgrove

"Nutrition of the Foal"
C. E. Hagyard

"Barker Foals"
J. S. M. Cosgrove

"Iso-Hemolytic Jaundice"
W. R. McGee

"Retained Meconium"
C. E. Hagyard

"General Principles in Radiology and Interpretation. II.
W. D. Carlson 187

"Equine Abdominal Surgery Technics"
F. J. Milne 205

"Equine Synovial Fluid"
C. E. Cornelius 215

Clinical Briefs: "Some Therapeutic Measures"
R. Copelan 223
" 'Tying-up' in Horses"
J. H. Fries 227

Directory of Program Participants 230

XUM

ERRATA

Page 21, Para. 2, Line 6 for 1,000 horses read 7,000 horses.

Page 29, Last Para., Line 4, for stage read stages.

Page 31, Editors Note, should read as follows:

As members present at the convention will recall, Dr. Snyder's contribution was essentially in the form of color motion pictures. It is regretted that it is beyond the ability of an editor to reproduce such a presentation in manuscript form, however in view of the excellence of the material presented and the interest shown by members, it was decided to print in its entirety the transcription of the remarks made by Dr. Snyder and those who participated in the discussion. This will at least help to recall to mind the salient points of a memorable presentation.

Page 34, Line 29, for SYNDER, read SNYDER.

Page 41, Para. 1, Line 9, for homo, read homo-,.

Page 47, Para. 2. Line 3, for exent read extent.

Page 72, Para. 2, Line 2, for treatmnt read treatment.

Page 89, Para. 2, Line 6, omit L after (1957).

Page 110, Para. 2, Line 2, somthing read something.

Page 136, Para. 2, Line 4, for gulconate, read gluconate.

Page 181, Para. 4, Line 9, for sensitizat on read sensitization.

Page 193, Line 4, for Fig. 15-19 (Fig. 13) read Fig. 15-59 (Fig. 13).

Page 197, Line 3, for obscrures read obscures.

Page 207, Line 3, for line read linen.

Page 228, Para. 7, Line 2, for calcuim read calcium.

XUM

QUARTER HORSE PRACTICE

C. T. Mason

Our way of life is changing. As one drives through any town, two new, common sights meet the eye. The first is the number of boat rigs and trailers; the second, almost as common and many hundredfold what it was ten years ago, is the number of horse trailers parked in American yards. The latter concerns us as veterinarians.

The American Quarter Horse Association was formed right here in Texas in 1940. No horse registry in the world has come so far, so fast. In one month of this year, hundreds more quarter horses were registered than in 1950. Last year, the Jockey Club registered over 12,000 thoroughbreds and the U. S. Trotting Horse Association registered over 1,000 horses; the Quarter Horse Association registered 46,000 horses — well over double the combined total of the other two associations.

Quarter horse practice is a pleasure because the veterinarian deals directly with a higher percentage of owners who train, breed or ride than is the case with other breeds of horses.

The demand for individual pleasure horses will continue to increase since there is a great possibility of a 30 hour work-week by 1970.

There are two main classes of quarter horse — the running and the working horse. The working horse class is divided into cutting, reining, roping, barrel racing, working cow horse and pole bending — showing the versatility of the breed. In addition, the dogging and hazing horses used in rodeos are usually of quarter horse breeding.

This talk will not be of a scientific nature but I hope it will help some of you in your practices. I obtained some of these tips from other practices and they have been of help to us.

Those who use dental floats realize that before long, the set-screws which hold the blades are frequently stripped or become rusted. We replace these with Allen-type screws and therefore blade changing, by means of an Allen wrench, is no longer a problem.

For general anesthesia (in castrations, hernia repair and other major surgery), we use 1 ounce of chloral hydrate, 6 grains of phenobarbital with enough water to make 500 cc of solution. The cost of this safe preparation is less than 30 cents per bottle. After more than 15 years of use, we have never left a horse on the ground. When tranquilizers are ineffective, this preparation may be used with safety for both horse and surgeon.

XUM

We use 1″ masking tape as a tail wrap before applying a blister or carrying out pregnancy and sterility examinations. If dropped, it does not unravel; nor does it require laundering as is the case with a tail or leg bandage.

In hard-to-settle mares and those manifesting silent heat, 250-500 cc of saline solution containing one or two Furacin squeejets* is instilled into the uterus. More than any other treatment, this has helped us to settle such mares. When nothing seems to be of value in settling some old mares, the application of a dental float to the teeth may bring results; in some wind suckers, 30-45 days after such treatment, the body weight will increase by as much as 150 pounds so that fat deposition balloons the vulvar lips and prevents further wind sucking. In some of these older mares, it is important to examine the mouth as well as the genital tract.

Years ago, Bill Carnes of Muskogee, Oklahoma, put me wise to the use of cotton picker knee pads. These are part of our standard surgical equipment especially for operations carried out with the animal down. For a couple of bucks, one can have real enjoyment while performing surgery!

Rather than waiting in the stall for a good mare to foal, we place an "intercom" in the stall with the speaker in the house. When the mare goes into labor, we can be in the stall in a matter of seconds. One can hear the mare chewing on her hay and there is no doubt about the sound when she goes into labor.

A good fly spray or repellent is applied before giving intravenous injections or working under horses. Incidentally, the horse will stand more quietly if the bottle of intravenous solution is held behind the head rather than beside the eye or ear where the animal can both see and hear the emptying of the bottle. Intravenous tubing is carried in a 1 pound ointment jar filled with a mild solution of Nolvasan**. Owners appreciate this point.

Horses suffering from "gravel" are frequently seen. After removing the shoe, but before curetting the lesion, Massengill's Topical Anesthetic is injected into the cavity. A horse-shoe nail with the tip bent at an angle of 90 degrees makes a suitable probe. Finally the cavity is filled with mastitis ointment and packed with cotton. The owner is instructed to soak the foot in Epsom salts solution, twice daily.

To cool out feet in cases of founder, an old inner tube wired at the bottom makes a satisfactory boot. If the occasional animal resents these boots, they should be removed as the exertion is not

*Furacin Solution Veterinary Squeejets is a product of Eaton Laboratories, Norwich, N. Y.

**Nolvasan (Brand of Chlorhexidine) is a product of Fort Dodge Laboratories, Fort Dodge, Iowa.

good for the animal. In these cases, we have the owner procure a child's plastic wading pool and add one or two hundred pounds of ice to the water in the pool. By this means, the adjacent stalls are not flooded with water.

Wire cuts and rope burns are scrubbed clean using a mild solution of Nolvasan and surgical soap. Dry sulfanilamide powder is then applied to the area and Scarlet Oil to which Lindane* has been added as a fly repellent (1 ounce to the gallon) is used. For lacerations and rope burns of the flexor aspects of the pastern where the horse flexes the fetlock and the scar tissue tends to maintain the state of contraction, it is recommended that the heels be kept trimmed or in more severe cases, the horse be shod with an extension toe.

For proud flesh we use a combination of equal parts of saturated solutions of copper sulfate and potassium permanganate. It is dispensed in one and one-half ounce size, plastic pink-eye spray bottles and when applied once daily, does a remarkable job. It is also of value for slow-healing sores. It is cheap and reliable in use.

In corrals where the stallions have closely cropped the grass, a deficiency of Vitamins A and D is often encountered. The stallions become so sore and want to go down when mounted or even when the saddle is placed on the back. A majority of these cases will clear up rapidly following administration of Vitamins A and D.

In colts with rickets, contracted tendons are likely to follow the swelling at the fetlock. If treated early with heavy doses of Vitamins A and D along with a good mineral preparation containing some magnesium, most of the conditions will resolve within 6-8 months. In addition, it is recommended that the feet be kept flat and the heels pared well down.

Every year, a number of cases of cellulitis are treated. The lesions are usually on the side of the ribs and along the area of the subcutaneous abdominal vessels. The condition arises from the rough use of spurs and also from a girth that is too tight. Reining and cutting horses that are spun, turned and spurred to a great extent are most commonly affected. For treatment, 250 cc of sodium iodide solution containing one and one-half grams of tetracycline is given intravenously and the areas punctured with the point of a scalpel. Following this, the lesion is rubbed briskly with white liniment.

Plastic obstetrical sleeves make excellent carrier bags for fecal specimens. Drop a sample with the name of the animal down

*Lindane LE 125 is a product of Thompson-Hayward Chemical Co., Kansas City, Mo.

XUM

the sleeve, tie a knot in the sleeve, put in another sample, tie off and so on. They are also useful for carrying post-mortem specimens to the office as they are handy and sterile.

In cutting, reining and some working horses, "stifled" animals are sometimes seen. In all such cases, the femur seems to be nearly perpendicular. In horses in which the femur slants at an angle of eighty degrees the condition is not seen since the epicondyles form a deeper groove for the patella. If I were judging a show and had to decide between two horses, I would definitely downgrade the one with the upright femur.

In over-worked horses where tetanic spasms prevent the removal of the bit from the mouth or where the horse cannot lift its feet over the threshold of the stall, 150 cc of calcium gluconate will usually effect a rapid cure. If this does not succeed, Robaxin* will usually relieve the spasms.

For strangles, our standard treatment is a large dose of triple sulfa and tetracycline given intravenously for two successive days.

Stable coughs are a frequent sequel to shows. For this condition, a double dose of Mixed Equine Bacterin, No. 1, and 15 cc of Otrhomin** are given subcutaneously in two or three locations. The owners are warned that the latter preparation will produce a temporary welt at the site of injection.

For the treatment of sweeney, two or three drops of one per cent silver nitrate solution are injected about an inch apart into the supraspinatus and infraspinatus muscles. The atrophied muscles are filled in and this can bring the owner an extra hundred or two hundred dollars for the horse or may help him in a halter class! A second series of injections is occasionally necessary.

When operating on cryptorchids, we employ general anesthesia. Two points should be stressed in this type of operation; (a) in placing the first suture in the internal inguinal ring, it is rather difficult to tie the first knot so we can make a honda or loop at the end of the suture material and after penetrating the lips of the internal ring, the needle is brought to the outside and passed through the loop on the other end of the suture material. The loop is then slid down to the internal ring and suturing continued; (b) when releasing a horse that has just undergone this operation, the animal should be placed on the side on which surgery has been performed. The reason for this is that the upper leg is always going to extend outwards and thus open the canal whereas the side that is down keeps the canal closed thus minimiz-

*Robaxin brand of Methocarbamol is a product of A. H. Robins Co., Inc., Richmond, Va.

**Otrhomin is a product of Weidnerit, Berlin-Charlottenburg, Germany.

ing the possibility of suture disruption and the passage of a loop of bowel down the canal.

EDITOR'S NOTE: At this point the speaker commented on some slides as follows:—

[Slide] Hoof grooving by hand, when considered advisable in cases of chronic laminitis can be very hard work and a high speed grinder is used to lighten the task. We use a ¼ by ½ inch oval burr or Nicholson rotary file. If the ground is wet it is advisable to earth the grinder and stand on a rubber sheet for obvious reasons! We use this same grinder with a dental burr for the repair of quarter cracks which do not appear to be as frequently encountered in the quarter horse as in the gaited and walking horse. The crack is undercut and filled with "sure-weld", a self-curing dental acrylic. Many of these animals go sound within a day.

[Slide] Reverting to hoof grooving, in addition to the "V" on the front of the hoof, parallel grooves are made all the way back to the heels. By this method we believe that contraction of the heel is prevented and the front of the os pedis is less likely to bring on the condition of dropped sole. The amount of relief following this type of grooving is often spectacular.

[Slide] This roping horse had been gored by a Brahma bull with a resulting laceration measuring some 18-20 inches long, extending into the abdominal cavity. Our plan was to tranquilize the horse and place a few stay sutures preparatory to administering general anesthesia, but evisceration took place and the protruded intestine had to be held in place by hand while anesthesia proceeded and suturing by layers was carried out.

[Slide] This slide illustrates a case of depressed fracture of the lacrimal bone over the maxillary sinus. About three inches of bone had been driven into the sinus and had to be pried loose with a spaying hook. If it had been left as it was, trouble would have followed. Removal of the eye was indicated and this was done after which the cavity was filled with sulfanilamide powder and the lids sutured together. Formerly we packed the cavity with gauze, but now by using sulfanilamide and not packing with gauze, complications are practically eliminated.

[Slide] This illustrates laceration of the gum and loosening of the incisor teeth in a colt. Formerly we went to considerable trouble to suture the gum and try to hold the teeth in position but after discussion with orthodontists we now remove the affected teeth and this prevents mis-alignment of the erupting permanent teeth.

[Slide] During a tornado, a horse will always turn away from the force of the wind and as a result flying debris may inflict severe wounds on the heavily muscled region of the quarter. Such

XUM

lacerations are not sutured but allowed to heal as open wounds. After cleansing with Septisol† and a mild solution of Nolvasan, sulfanilamide powder is liberally applied followed by a coating of Scarlet Oil containing Lindane.

[Slide] In purpura hemorrhagica, swelling of the head is not necessarily present. In addition to the swelling of the legs and lower abdomen, a purple coloration of the nasal septum is often noted in the early stages; if resembles the color noted in Infectious Bovine Rhinotracheitis. For treatment, 8 cc of formaldehyde in 350 cc of distilled water is given intravenously. Large doses of Vitamin B complex and ascorbic acid are also given. Although many practitioners use steroids, we prefer not to administer them on account of their tendency to slow the healing process.

[Slide] This shows the repair of a fractured leg in a colt. Rather than work on the ground, we lifted this animal on to a picnic table. The plaster of Paris cast is reinforced with strips of 20 gauge sheet iron.

[Slide] To obtain the amount of extension necessary to set a fracture, regular or horse shoe nails are driven through the hoof to hold a loop of baling wire. To this loop is attached the fence-stretcher or lariat.

[Slide] We encounter mouldy corn poisoning more frequently in wet than dry years. In addition, poisoning seems to result not from corn produced in the current year but from that which has been held over from the previous season. Fermentation and the change of weather seem to precipitate toxicity. For treatment, 125-250 cc of phenyl-mercuric nitrate is administered intravenously along with small doses of Istizin* and Lentin** to effect purgation.

[Slide] In cases of snake-bite, we pepper the swollen area with the point of a scalpel and then massage briskly with camphorated oil. The intravenous injection of 100-150 cc of Havidote*** seems to help these cases.

[Slide] Following the removal of neoplastic tissue from the penis and sheath of a stallion, deviation of the penis may follow. To prevent this, the horse is kept under the effect of tranquilizers for about five days after surgery. The extended penis is thus less likely to develop a curvature.

---

†Septisol (brand, antiseptic liquid soap) is a product of Vestal Laboratories, Inc., St. Louis, Missouri.

---

*Istizin (Dihydroxyanthraquinone) is a product of Winthrop Laboratories, New York.
**Lentin (Brand of Carbachol Injection, U.S.P.) is a product of Merck & Co., Inc., Rahway, N. J.

***Havidote is a product of Haver-Lockhart Laboratories, Kansas City, Missouri.

[Slide] This slide shows an inner tube securely fastened by a rope or web strap at the top. At the bottom, is attached the lead shank of the young, unbroken colt. This in many cases will prevent fractures or dislocations of the cervical vertebrae as well as prevent the destruction or tearing up of many halters. (Fig. 1).

[Slide] This animal, suffering from osteomyelitis of the tuber coxae, had been treated for several months by establishing drainage. The only way to correct the condition is to anesthetize the animal and chisel out the diseased bone.

[Slide] A couple of years ago, we treated several cases of infected guttural pouches following strangles, shipping colds, etc. We inject the pouches with Varizyme* or Chymolase**, entering the pouch under the wing of the atlas and a day or two later establish drainage through Viborg's triangle. A mild antiseptic solution is used to flush the pouches.

[Slide] For the surgical repair of umbilical hernia we used to wait until the animal was six months old. Now we prefer to operate as soon as possible.

[Slide] This two year old horse was not recognized by the owner as a hermaphrodite until we had cast him.

[Slide] Since equine infectious anemia is present in our area we autoclave all syringes in O.K. sterilization bags manufactured by Propper Manufacturing Co. Owners appreciate this aspect of preventive medicine.

In some cases, nasal hemorrhage follows the passage of a stomach tube, so we prefer not to use this method of drug administration in certain locations and thus minimize the chances of spreading infection.

We advise owners in these areas to make use of a pressure cooker for sterilizing needles and syringes. To demonstrate the seriousness of the condition to the owner, we draw some blood into a tube and hold it against the car windshield with the help of the wiper. The owner can see for himself the abnormal nature of the blood although it is pointed out to him that every animal with disintegrating cells and a low red-cell count does not necessarily indicate swamp fever.

[Slide] This port-a-fold stool acts as a useful instrument table and also as a chair for the veterinarian while waiting for the owner to get his animals up or for the colic case to get better.

QUESTION: What was the treatment for mouldy corn poisoning?

---

*Varizyme is a product of American Cyanamid Co., Princeton, N. Y.
**Chymolase is a product of The Warren-Teed Products Co., Columbus, Ohio.

XUM

DR. MASON: Phenylmercuric nitrate (1:1500 solution), 125-250 cc given intravenously. Then we purge them with Istizin and Lentin. The quicker we can eliminate the material in the bowel, the less will be absorbed.

DR. C. H. REID: I would like you to repeat the formula you use for proud flesh.

DR. MASON: A concentrated solution of copper sulfate and concentrated solution of potassium permanganate, equal parts.

DR. REID: Saturated?

DR. MASON: Saturated solution, yes, equal parts. Put it in a glass or plastic container because it will erode a tin container.

DR. REID: Just an idea! Having lived in the days of corn-stalk poisoning, if you take the corn you are going to feed and put it in a pail of water and let all the debris come off the top, and then feed the corn, you won't have this trouble.

DR. MASON: That is worth noting. Let me ask, have you ever had cases with this year's corn?

DR. REID: No.

DR. MASON: I don't believe I have ever found any. I think possibly the fermentation and moulding has something to do with it.

QUESTION: You didn't mention your treatment for purpura. Do you have one?

DR. MASON: We revert to the old treatment of 6 to 8 cc of formaldehyde in 350 cc of water. Give this slowly because it "shakes them up" a little. This will very definitely help. We use a lot of B complex and ascorbic acid. We stay away from the steroids. I know many people are using steroids, but I believe you will slow up healing enough to where it gets dangerous by using the steroids.

QUESTION: What do you feel your formaldehyde is benefiting?

DR. MASON: I don't know, but I have seen them with a head this big, which "shrinks" down today, and is back to normal by tomorrow. Results are what we are looking for.

CHAIRMAN REED: Who makes the high speed grinder you are using?

DR. MASON: The one I am using is a Craftsman by Sears Roebuck.

DR. C. H. REID: Regarding purpura, I am glad you brought out the point that you don't always see the typical case. Sometimes in our practice the lesions have been confined to the two

hindlegs, and one will clean up real fast but the other will slough. Want to talk about that?

DR. MASON: I believe you just about covered it. So frequently, at least in our case, we will see hemorrhages on the gums, nasal septum and conjunctiva. There will be enough pressure in some of these areas to give rise to extensive sloughing.

QUESTION: What was your dosage of Robaxin in muscle spasm?

DR. MASON: Usually about 10 cc.

QUESTION: Intravenously?

DR. MASON: Yes.

QUESTION: You mentioned leptospirosis. Are you treating this condition? If so, what are you using? What kind of results, and how long treatment?

DR. MASON: We are treating it, but with what results, I don't know. Many of these mares just lose their foals, go on eating and yet still show a high titer. We are using streptomycin-penicillin on them, but I do not know what is going to happen when we try to get them settled next year.

DR. R. S. JACKSON: Regarding the settling of these mares next year, I doubt if you will have any trouble at all. We have seen quite a bit of it out there, and my experience has been that the following year, or even that year in which you are breeding them, you have little or no trouble in settling those mares.

DR. MASON: I will say this; we are vaccinating against L. ictero-hemorrhagia and L. canicola, in those cases where the titers are high. I don't know whether vaccination against L. pomona would be of value.

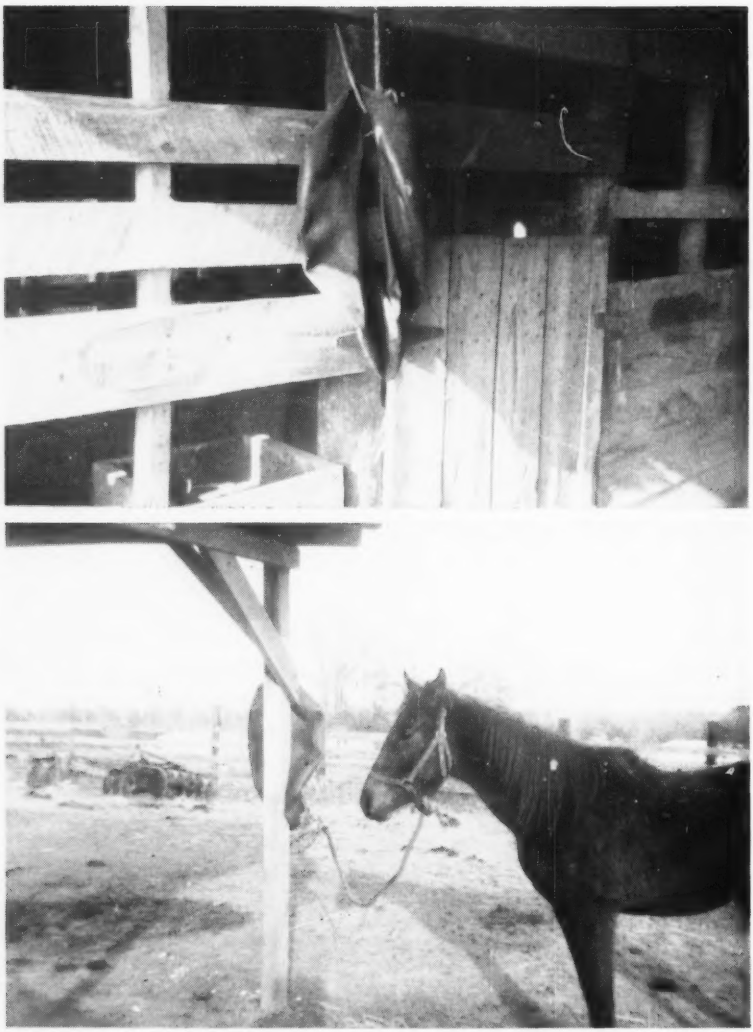
QUESTION: How often are you vaccinating them?

DR. MASON: Twice, and repeating in six months.

DR. BARSALEAU: In leptospirosis, are you running into a lot of eye lesions—iridocyclitis? If so, what is your treatment?

DR. MASON: Yes, that is something that we do run into. Dr. Bill Magrane is a lot more capable of talking about that than I am. One thing that helped us is to see the animal between the acute stage of periodic ophthalmia, when the owner knows there is something wrong with the horse's vision. The Merck Manual mentions the use of fluorescein stain, injected intravenously. It will surprise you just how fast some of that drug will come through on some of these. You can, I believe, forecast which eye stands a chance of coming back to normal after an attack. When you use this fluorescein stain, keep the horse out of the sunlight. I left some of them out in the sunlight and some developed photosensitization. Put them up for at least a week where they don't get into light, or trouble will result.

XUM



MASON; Fig. 1 Tethering of a young, unbroken colt. Note that the inner tube is placed sufficiently high up to prevent a foot being caught should the colt rear up.

THE SURGICAL HANDLING OF TISSUES

CLIFFORD SNYDER

EDITOR'S NOTE: As members present at the convention will recall, Dr. Snyder's contribution was essentially in the form of color motion pictures. It is regretted that it is beyond the ability of an editor to reproduce such a presentation in manuscript form, however in view of the excellence of the material presented and the interest shown by members, it was decided to print in its entirety the transcription of the remarks made by Dr. Snyder and those who participated in the discussion. This will at least help to recall to mind the salient points of a memorable presentation.

CHAIRMAN REED: Gentlemen, this afternoon we are most fortunate in being able to have with us one of the finest plastic surgeons in the world. This man is Assistant Professor of Surgery in the School of Medicine at the University of Miami. He is a Diplomate of the American Board of General Surgery and a Diplomate of the American Board of Plastic Surgery. He is a winner of the International Plastic Surgeon's Award. He is a Fellow of the American College of Surgeons. He is the present Secretary of the American Society of Plastic Surgery, and President-elect of the Southeastern Society of Plastic Surgery. On top of that he is a real good surgeon; I watched him. (Laughter). It has been my privilege to watch this man do some fantastic things.

This afternoon he will present three movies. The first will be research with surgical technics; the second will be instrumentation, and the third, clinical material.

After the presentation of these movies, we will have an informal session, and Dr. Snyder will answer questions regarding plastic and reconstructive surgery. Gentlemen, it gives me great pleasure to present Dr. Clifford Snyder. (Applause).

DR. CLIFFORD SNYDER: Dr. Reed, Ladies and Gentlemen: I am embarrassed but I am happy to be here. I am embarrassed because I really feel that your Program Chairman and your Program Committee could have done much better in their choice of speaker today. But I am happy for a number of reasons; one is just to be with you. Another is that I was born in this city, just down the street, about fifty years ago. About ten years ago I gave a paper in this same hotel before an organization called the Texas Surgical Society, which is quite an esteemed organization in this state. When I was introduced, the doctor who did the introducing was the son of the man who delivered me. (Laughter). He introduced me by saying that, when his daddy practiced medicine, in the days of the patients' paying with vegetables and meat, and so forth, I was a $15 baby. (Laughter).

Now, you are well acquainted with figures like this because you talk of newborn colts, $50,000, $80,000, and so forth, but not $15 babies. (Laughter).

XUM

The veterinarian has become an integral part of my life. He comes into my office whenever he desires, and I feel welcome in his office. He comes into my operating room whenever he wishes, and I make myself familiar in his operating room, as Dr. Reed has told you. He eats in my home, and sometimes I have the pleasure of eating in his. I have slept with veterinarians (laughter) in the Everglades swamps, in the Wyoming mountains and also in the Texas valley. I have operated on veterinarians, and they have operated on my animals (laughter) and one time on me. This was in regard to a snakebite. At a recent meeting, we counted thirty-two miserable, unfortunate veterinarians on whom I had operated. At least I was able to follow my complications. (Laughter).

So, you see, I do feel that I am amongst friends, though I remain somewhat suspicious of you. You always act so ignorant, at least in my company, but before we part company, you veterinarians are always answering my questions instead of me answering yours. I am proud of veterinary medicine in our country and I am equally proud that I am a part of it.

This first movie has never been shown before to an audience. The veterinarian, Dr. Knowles, who is associated with me on this project, and I reviewed it on Thursday at lunch time. I may mention that the end of the movie has not been completed because we want to show our dog at a later stage and there has not been enough time lapse to illustrate our results. This is known as soft tissue handling operative technic. It is the amputation of a dog's leg and replantation. This color film is in sound but if any of you have a question during the movie, or immediately after, while it is being re-wound, I will be very happy to attempt an answer.

(Motion picture, "Leg Replantation" with additional commentary by the speaker)

We had to furnish all of our own money originally and our instruments were not the best, but now we have a small grant and better equipment.

The machine that you now see has many names, including the heart-lung machine and pump oxygenator.

We are now using homogenous bone grafts for this intramedullary bone fixation procedure. We take these bone grafts out of another dog and use them as nails. This has never been done before as far as we know.

We like wire in closing the skin because the animal then does not bite the wound. The wire ends nip his or her tongue and they do not tend to injure the wound.

While he is rewinding the film, I would like to say that we do have one dog alive, which we did in May, 1960, about nineteen

months ago. This dog has a viable extremity but he is unable to feel a pin prick in his foot pad; in other words, he does not have sensation.

What is the object of this procedure? Why cut a dog's leg off and put it back on? A number of years ago there was a child in an automobile accident on the highway outside of Miami, called the Tamiami Trail, between Tampa and Miami. The child was brought into the emergency room with the arm amputated, slightly macerated at the end where the amputation went through the extremity. At the time when I saw the child, I desired to replace the extremity, knowing that, if it could be done, the youngster would not have to use a prosthesis afterwards. The youngster was in shock, due to loss of blood. Surgically, it would have been a difficult procedure because we had never done one before. It would have taken six or eight hours to approximate the various vital structures in the extremity. Therefore, the child was a surgical risk if such a procedure was attempted. Regardless, the case was unforgettable. Since then I had attempted to replant animals' limbs without success. Only did success arrive when Dr. Robert Knowles and I pooled our resources to achieve the desired result. We are now ready for such a catastrophy to happen and we think we will be able to save the next one.

Because of this research project, I felt it necessary to find out what other countries were doing. You and I have in the past read in the newspapers, magazines, and so forth, where the Soviets had replanted limbs, and also many other wonderful things they had been doing. So, I felt it necessary to go to the Soviet Union to see their results. I examined their research centers. They were very hospitable people. Two men were exceptionally fine, Vladimir Demikof who has done the two-headed dog and also the two-heart operation, and Lapchinsky who has replanted limbs. Lapchinsky was able to restore sensation and motion. We have not been able to achieve this because our amputations are high. It can be achieved if the amputation is below the point where the neuro-vascular bundle comes into the muscle. About three years ago, a paper was given by one of the co-authors of this movie, and it was proven that a nerve transplant would work, though we had been taught differently. So, we have made progress.

The next movie concerns the Soviet vascular and nerve anastomotic machine. This is a machine that we bought from the Russians and have used many times, and now we think we have one in the making that is better. This instrument was not invented by the Soviet research men, though our newspapers did give them such credit. It was originated by a Hungarian, and the Russians adopted and perfected the technic. It is a beautiful machine and will be clearly demonstrated here. This color film is

XUM

also in sound and I do not think I will need to say very much about it. By the way, we have the movie started in our post-operative dog, and maybe two years from now when you meet in Miami Beach*, maybe we can show you these post-operative pictures.

(Motion picture on vascular anastomotic instrument).

While the projectionist is rewinding this movie and preparing the last one, I will reiterate that now you have seen 1. soft tissue handling and 2. you have seen development of new, mechanized surgery. Now this last, the third portion of the program, is on the clinical use of these technics.

The case in this movie is that of a lady golfer. She was entertaining someone who was of great prominence in our country. During this time, the washing machine was functioning in the anteroom. The lady who was taking care of the washing machine did not speak English and could not understand English too well, so, the hostess went into the anteroom to turn off the washing machine. She used a terrycloth towel to stop the rotary motion of the machine and, in so doing, the towel got caught in the machine and carried her thumb into the blades, which resulted in an avulsion amputation of the thumb.

An attempt to replant the thumb was not undertaken by the physician. The result was a hand without a thumb. The thumb is nearly 50% of a hand and important to most people. We transplanted the ring finger to furnish the function of the thumb. We have now done six cases and they function exceptionally well.

DR. W. F. JACKSON: What is the technic of suturing the nerves together — try to get it outside?

DR. SYNDER: The idea in bringing the severed ends of a nerve into apposition is to not injure the neurofibrils, that portion of the nerve that is on the inside. So the technic used is just to suture the epineurium, or the covering of the nerve. Heretofore we have been taught, and we teach, that a peripheral nerve may not regenerate. I am beginning to doubt this now since Lanchinsky has proven that this may not be so. I now feel that if the surgeon utilizes a high-powered lens while very carefully anastomosing the nerve, it may regenerate. The ends of the little arteriole that goes along the nerve is the indicator of how the nerve should be brought together and when this is lined up, the nerve should then be approximated.

CHAIRMAN REED: Do you use a microscope for your operation?

*Editor's Note: The venue has been altered to Lexington, Kentucky.

DR. SNYDER: No, we use binocular lens which magnify.

CHAIRMAN REED: You use 5 power?

DR. SNYDER: We use 5 power which works nicely.

QUESTION: I know that one of the difficulties in anastomosing blood vessels is clotting in the vessel. What precautions do you take to prevent this?

DR. SNYDER: We sometimes heparinize our dog but not always.

CHAIRMAN REED: Systemically?

DR. SNYDER: Yes. We use the blood in our oxygenator, the heart-lung machine, from that dog, but it is not necessary. We take this transfusion from the dog the day before, or that morning, and we utilize that same blood in our pump oxygenator to treat that limb, while it is off.

By the way, these limbs can be kept 24 hours off the host animal. This is good, because if someone has amputated an arm or a leg somewhere else, and it takes time to get to you, there still may be a chance for survival. We feel that the secret is the pump oxygenator. It does not have to be of the type seen in the movie. We built this one ourselves. It can be smaller and it can be very expensive. This one cost us around $1,100 when we finally finished with it. We feel that the perfusion, getting the arterial and venous tree free of blood clots is the answer to the retaking of a limb. We feel it can be done successfully, with careful work. If we encounter such an injury, this veterinarian will be working right with me in the operating room.

DR. CHURCHILL: In amputations, in what percentage of cases do neuromas develop, and what do you think about them?

DR. SNYDER: I do not know the percentage of cases of neuromas developing in the animal. They develop 100 per cent in the human. (Laughter). There isn't any question about it. We expected such a question and, by the way, this wasn't pointed by Ed. I just met Ed today. It just so happens I have some slides. (Laughter). The procedure that we have done for neuromas is something a little bit different from that you have seen previously.

[Slide] There are many, many treatments for neuromas. This is a man's forearm that has been amputated below the elbow. I have marked three neuromas as you see on the slide.

This man has had many treatments for this amputated forearm. The last procedure done was to shorten the bone and put some fat pads down to cover the bone, with the idea that the bone was causing his trouble, but this was not the trouble; it was neuromas.

XUM

A veterinarian, who today is President of this Equine Association, gave me this idea on how to treat neuromas.

[Slide] This is at the operating room. My left index finger points to one of the neuromas. The other two hemostats are on the other neuromas. We have exposed them, showing you how large they really are, and they are extremely tender and very painful.

[Slide] Dr. Teigland said, "Cliff, in the treatment of your neuromas, have you ever tried to amputate, remove the neuroma and utilize the adjacent vein to bring the end of the nerve up through?"

Since the nerve has a tendency to grow but doesn't know where to grow — it grows in whirls, forming a knob, but if you put it into something like a sheath, along which it can grow, it does not form a neuroma.

This slide shows the vein being pulled out with a hemostat, to lengthen it. I have introduced a needle up high in the vein, brought it out and attached the end of the nerve to it.

I have taken a straight needle and gone through this vein right there and come out here. Then I used what we call a Bunnell type of suture in this nerve.

[Slide] We cut the vein off and bring the nerve right up into the vein. I did two of these nerves just to check this technic that Dr. Teigland suggested and the other one was imbedded into muscle.

Today, which is now ten months later, the two that I put in the veins have not given this man any trouble whatsoever, but the one I put into the muscle is giving him all kinds of trouble. He does not know why; I have not said much to him (laughter), but I will, sooner or later, go back and take that other one, the radial nerve, and do the same, which will give me an opportunity to check these others.

Dr. Reed and I have spent much time discussing technics on neuromas, because you fellows have a lot of trouble with the volar digital nerve in the horse's anterior limb. We did put a horse on his operating table, and ran through some different procedures. I have got some other ideas now, since these two veterinarians have stimulated me, and I honestly feel that we are going to find the answer. Bill has injected some of the nerve endings, amputated the neuroma with various solutions, one of them being a sclerosing agent. He is following this along now to see how his results are. There are at least 150 different ways of treating a neuroma.

Do you want to comment on this, Bill?

CHAIRMAN REED: I will comment to this extent: Last winter, Cliff and I did some where we folded the nerve back; we im-

planted the nerve. I thought this might work fairly well. We were trying to apply the same principle, burying it in the vein. I did, I would say, 30 or 40 of them in different ways. I would make a little false tract, take a Kelly forceps, take a little tack up on the subcutaneous tissue and implant the nerve by running a double needled suture through the nerve and tying it on the outside. At the end of about a week or ten days, I would take it off. I thought this would work very well, as it looked promising. I would say this, the neuroma was much slower in forming but it ultimately formed. It was a disapointing result. Then we started the other things Cliff mentioned here.

DR. SNYDER: But the main idea comes back to the same thing; it is teamwork, and it is teamwork between the medical doctor and the veterinarian. When I have something in mind that I want to try, I call one of my veterinary friends. He finds the animal, small or large, and we work it out together.

Sometime next year, because of the research they helped me to do, I will be going to India to construct a bladder for a very important individual over there who was born without a bladder. I would not be able to do this if the veterinarians had not helped me and stimulated me with ideas. I have heard the term "He is a horse doctor" and I guess I have been called many terms by patients, too. (Laughter). I would rather have you be called a doctor with horse sense, because when we speak of horse sense, we feel that there is someone who has got the old common mind he needs.

[Motion picture] This is not in sound and this film has not been edited, so I will have to explain things. This is the lady who lost her thumb. She is a professional golfer and did not want any thumb to be made from her abdominal wall, with a bone graft stuck in, or anythink like that. She wanted a finger, that looked like a nice finger so that when she played bridge, the people wouldn't criticize it.

With her permission, I decided to use the ring finger of the right hand. You must be careful in dissecting the artery, the vein and the nerve. You may sever a volar digital artery and you will have another one or collaterals, that will carry the blood down, but if you sever or lose a vein, then you only have one left and, if it becomes thrombosed, the finger is lost.

Here you see we are dissecting the tendon sheath to expose the tendons. We left these intact, but we must move them. The extensor tendon is the one that is severed. It is re-anatomosed to the thumb extensor later. In industrial surgery, at least in my state, the thumb is 50 per cent of the hand. In other words, if you lose a thumb, you have lost 50 per cent of the hand, though I do not grade it as such. That is what the Florida Industrial Commission states.

XUM

With this in mind, the thumb is the most important finger on the hand. You now see the extensor tendon, the one on the back of the finger. Now, you see the little connecting branches of the vein. They go from one finger to another. They must be severed because you can't transfer the finger unless they are severed. This on is severed and tied. You do not use the electrocoagulation apparatus here to stop the bleeding in this vessel because you may injure the main course of the venous return; hence you tie off carefully.

QUESTION: Use a tourniquet?

DR. SNYDER: Yes, in nearly all the surgery I do on the hand I use a tourniquet. The pressure of it is evenly distributed, so that there is no injury to the soft tissues when it is applied. On the question of how long to leave it on, I have left a tourniquet on an extremity two and one-half hours, without damaging it.

Here we are severing the phalangeal bone.

We are ready to transfer this finger over and make a thumb out of it. The reason that we chose this finger on the right hand, the ring finger, is because people will not notice its absence. Later on you can draw your own conclusions as to the result.

This shows the preparation of the recipient site. If this nad been a male, or a lady who works in a factory, or someone who was not cosmetically minded, the space between this amputated thumb and the index finger, which we call web space, could be lengthened or deepened, and an individual could have a nice stump that the index finger would be able to touch, and real good function accomplished.

I was not going to make the incision of cutting the palmar skin at first but I got concerned about putting the finger underneath the skin, instead of bringing it across. This way I could watch the blood supply better, so I went ahead. I did one about two weeks ago, in which I just brought it right underneath.

You now see us cutting the stump of this metacarpal bone from the thumb, so we will get a raw surface of bone; the two bony ends will come together and unite. I take a portion of this bony stump, as well as that of the finger that we remove, and I use that as a bone peg. This is where Dr. Knowles and I got our idea about using a bone peg as an intramedullary nail. I have asked a number of orthopedic surgeons if a real long bone peg has been used as an intramedullary nail. When we speak of an intramedullary nail, we speak of one that is four or five inches long. Everyone I have spoken to has said he has never used it, though they have used it as I am using it here now, as small, short areas.

The first one of these that you do, takes about three and one-half or four hours. After you have done a couple, your technic is

worked out, and you amaze yourself as to the short time required to do one.

Here we are cleaning the bone off, getting ready to utilize it as a bone peg. We cut it to shape with an electric saw.

This is completely united now. We have x-rays of it. This lady is back on her golf circuit again.

By the way, you folks are the first ones who have seen these three films. I would like you to criticize them so we can improve them.

We are reaming the cancellous part of the bone, the canal, to fit our bone peg. There is the bone peg. Notice the flexor tendons come down the middle portion of the palm, and then they go to nearly a right angle toward the thumb, which is very good, because then that gives the action that we want on this finger, to make it work like a thumb. We have fixed this intramedullary bone peg with a Kirschner wire.

CHAIRMAN REED: How long do you leave it in?

DR. SNYDER: The question was asked, "How long do you leave the Kirschner wire in?" You treat this as a fractured bone — six to eight weeks. It is amazing how a finger looks more like a thumb when you get it over in that area. (Laughter).

The audience as a whole will be interested to learn that the American Medical Association and the American Veterinary Medical Association are now beginning to tie themselves to one another in regard to research. This was introduced at one of the latest meetings of the governing body of our medical group, and we expect to see these ties more closely knitted, which I think is a wonderful thing, because I do not know anything about the animal, whereas the veterinarian can help me along these lines. The veterinarian does not know as much as most doctors do about the human, so I can help him a little bit. It is a synergistic thing, and if we work together, I feel that the entire profession of medicine will be better.

I would like to suggest that, when you men get back to your home areas, if you do not already have a good medical friend, introduce yourself to one and start working with him, and get ideas from him; he will ask you for ideas.

Again, we use wire. I like wire on areas where I am going to leave the suture in for quite sometime. I am under the impression there is little reaction to wire, and that it does not act as a wick and carry perspiration down into the wound. This is an extensor tendon we are going to attach. It is a long extensor to the thumb, and we attach this now so that we have extensor function. This is the one we severed to get the finger over.

A lot of this should be deleted. You know, when you edit one of these films, you take out all of your bad technic and show only your good technic!

XUM

This is #36 stainless steel monofilament wire. I do not know how you men in veterinary medicine feel about elevation of a post-operative wound. We, in our field, are under the impression that if you get good lymphatic drainage and you can elevate a part, you get better healing. If there is edema in an area, you get poor healing.

I show the patient about six months later. There weren't any skin grafts or skin flaps, or anything like that, just transplantation of one digit to another. You cannot see too well where we have removed the finger. See, the web spaces are pretty nearly the same. She has good power.

If there are any questions, I will be glad to answer them.

QUESTION: You talked about electrocoagulation and cautery. What type of unit do you use, and do you use this for any primary incision?

DR. SNYDER: When I speak of electrocoagulation, we in the field of medicine are taught various types. We are taught coagulation, cauterization, fulguration, and there are others. In the office, if I need it for lesions on the face, and things like that, I use just the little old hyfrecator. In the operating room, where we are doing deep work, I use the bipolar method. You put a plate on one side of the patient, and the needle is the other pole. That is the bipolar method, instead of unipolar. That is electrocoagulation; it also has a cautery machine attached to it in the hospital. I do not think you need bipolar in your work; I think the small hyfrecator is sufficient. I use it quite a bit, but when I am training someone, a medical student or intern or resident, I first make him learn all the technics of tying a slip-knot, square-knot, all the one-handed and two-handed instrument ties, before I let him use this. Then he can use the easy way, but he must know the hard way first.

QUESTION: If one has a good cow and wants to take teeth from some old cull and put it in the good cow, how about this heterologous transplant? The physiologists said we could not do it; it might take, and six weeks later drop off, and they were right. What is your experience there?

DR. SNYDER: I would like to answer that in a long way. Yesterday the chest surgeon, better known to you as a heart surgeon, earned his bouquet of flowers. In the past decade, heart surgery has really blossomed out. I am under the impression that this decade, the 1960's, will be the decade in which transplantation of tissues will blossom. Whether we definitely will find an answer or not, neither I nor anyone else will be able to say this afternoon. I am under the impression that before this decade is over, we will be able to transplant a limb. That is a very harsh statement, but I cannot help believe it, knowing what I do and what I have reviewed in the past.

In answer to the Doctor's question, there are three types of transplants: the autogenous, that is, taking one of my teeth out of one side of my jaw and putting it in the other. Because you took it from me and you gave it back to me—autogenous. Homologous is when you take it off my friend, someone that is a man or a lady; in other words, my species. If you take it off my family and give it to me, it is an isograft. If you take it off a monkey and give it to me, it is called a heterograft. So, we have the auto-, homo, iso- and heterografts.

In this case in which the tooth was removed from one animal and placed in another, we do not believe today that it will work. In at least 99 per cent of the cases, this is so, but there is the odd case where it will work, and we do not know why.

I can demonstrate by a case of a little Mexican girl who was burned badly over 90 per cent of the body. Such cases nearly all die. We utilized negro skin, white skin, and Mexican skin on this youngster. Surprisingly, the negro skin and some of the white skin took and lived. We use the word "take". When you put a skin graft on, it takes, looks good, and so forth, but later on we lose it; it either sloughs off or it is autolyzed. But it took on this youngster and lived. We took sections of it afterwards, and you could see the growth of the melanin into the host tissue. We do not know why but we feel that the youngster's body was at such low ebb, such poor condition, that the antibodies were not strong enough to get rid of this; therefore, it took.

That leads to the man who is now the latest Nobel prize-winner in the field of experimental surgery, Medawar of London. He is a laureate today because of his work along this line of antigen-antibody reaction. The only thing that is holding us back today on transplantation of any tissue, is the so-called antigen-antibody reaction. They have used x-rays to knock it down; have taken spleens out. They have done various things to correct this reaction. We do not know why we have such a reaction. We have tried to captivate the various antibodies of all types, captivate them and hold them in the blood stream on certain cells, to keep these antibodies from reacting against our antigen, so that the antigen will take. This has been achieved to some degree, but it has not been perfected. There is the occasional case where the heterograft may take. A homograft will. I have had homografts in my own practice take—five or six skin grafts. They were of different color and looked funny. I wonder why they took and lived, when they should not have done so. I do not know. Blood groups may be an answer, but it hasn't been so far. The subtypes have been delved into right down to the nth degree. I just do not know the answer, and no one else does either.

In February, the New York Academy of Science meets in New York City, and we are going to have some arguments about

XUM

this. The Soviet boys are coming over. There is a man out in California now who has been doing some replantation of rat limbs. I know a lot will be coming out of this next meeting. I am sorry I cannot answer the question any better.

QUESTION: I wonder if anything has been done as far as replacing pigment is concerned. We have a real problem in horses, with loss of pigment due to scars. This is very important as far as show animals are concerned.

DR. SNYDER: I would like to shake your hand and let you join my family, too. We have the same trouble. We have the negro who gets burned and has white skin, and when the white man gets burned he gets a colored skin. Not only that, when we use a skin graft from one part of the body and put it on another part, sometimes it becomes pigmented. We think this may be due to lack of oxygen in the tissue, and that this is the reason it becomes pigmented. Replacing pigment in skin has not been perfected today. Tattooing has been used. Islands of skin have been placed in areas, hoping they will grow, but just the opposite happens; they shrink, and scar tissue sets up amongst them. So, we do not use too many pinch or island grafts. No, sir, we do not have a way today.

I wish somebody would ask me something I can answer in a positive way. I feel embarrassed about all these negatives.

CHAIRMAN REED: I will ask you a question, Cliff. What are the most important considerations, in your opinion, in the handling of soft tissue?

DR. SNYDER: I did not have time to elaborate on the movies, but I think the answer, in handling soft tissue injuries, is first to handle as many as you can with your fingers, instead of with large hemostats, Oschner and thumb forceps.

I think you should respect these tissues as you do your own, as if they were on your body or that of your little girl or your little boy. I think, if you respect the tissue, you will handle it more carefully. We, who are in plastic surgery, cannot afford scars. The general surgeon can get a big scar and the patient says, "Look what they have done to me. I really have something here, and I am proud of it." (Laughter). The poor old plastic surgeon is the one who has to take it off, and get a real small scar. I think the secret in handling soft tissues is not to macerate them, to treat them like they were your own.

You know, I feel that is the way it is in the entire field of surgery, Bill, that if we handle every case, whether it is a pig, a cow, horse, mule or what, as if it was our very own, that it belonged to us, we would make very few errors. That is the way we handle our little babies or our children, as if they were our own. If the doctor handles a case that way, he is going to take pretty good care of it. That is the reason for our good results.

COMMON INJURIES OF THE EYE

W. G. Magrane

Let me assure you at the outset that although I am happy to be with you today, and feel comfortable about being in the right church, I am a bit apprehensive about this being my pew. As most of you know, my work is primarily with the dog, and concerns the horse only when consultation and treatment of eye diseases becomes necessary.

Were it not for the fact that the eyes of man, the dog, the horse and other animals, for that matter, are essentially the same as regards anatomical structures and are subjected to the same injuries and diseases, I certainly would not have accepted this invitation to discuss the subject with you.

My first paper today will concern itself with some common injuries—a few common conditions applicable to the eye of any of these species and first I shall spend a few moments reviewing the anatomy with you.

(Several slides illustrating basic optical anatomy were discussed at this time — Figs. 1 and 2)

Concerning the methods of examination, in the first place, I am certain that, to conduct a proper examination, whether it be on the dog, horse or human, it should be conducted in a semi-darkened to darkened area, with a focal light source, such as a penlight or otoscope light. Some form of magnification is desirable, such as a binocular loop, although, in the case of the horse's eye, this is not quite as necessary as it would be in the smaller eye. I do not go along with that which I have read on a number of occasions about examination of the eye in sunlight. In the first place, in a soundness examination, in examining the eye in sunlight, one will not see much beyond the cornea, the lids and the bulbar conjunctiva. It is necessary to have contrast in the form of semi-darkness to darkness, and a focal light source, in order to study the anterior chamber, the pupil, the iris, and the lens, with which, of course, you are also greatly concerned in an examination for soundness or examination for disease processes.

We must get in the habit of thinking and looking beyond the conjunctiva and the cornea. We should follow a regular procedure in examination, looking first at the upper and lower lids, the palpebral conjunctiva of the upper and lower lids, in so far as that is possible, the bulbar conjunctiva of the globe and then the cornea, looking for foreign bodies, opacities, blood vessels; then looking into the anterior chamber for exudates; then the pu-

XUM

pil whether it contracts and dilates properly in the presence of light stimuli; the iris of the eye itself. Then, the lens, immediately behind the pupil should be examined. This can be conducted, of course, by gross examination or preferably with some form of magnification such as a binocular loop.

When it is necessary to examine further the deeper structures of the eye, we must resort to the use of the ophthalmoscope. The ophthalmoscope can also be used for the external examination of the eye, although essentially it is an instrument for examination of the fundus. If it is used on the plus 20 lens, we can examine the cornea very nicely with this magnification and then, proceeding with less plus lens down to O, examine the anterior chamber, the iris, the lens, the vitreous and down to the fundus, all with the use of that one instrument, the ophthalmoscope. So, probably from the standpoint of any one particular piece of equipment, this would be the most valuable. A penlight and binocular loop, however, will certainly enable one to make a good gross examination through to the lens of the eye.

Often on consultation cases, I have noted pus and blood in the anterior chamber, (Fig. 3). I have noted subluxation and luxation of the lens (Fig. 4) or foreign bodies of the conjunctiva or cornea. All of these things had been missed because the examination was not conducted in proper surroundings, and with proper equipment. Witn the proper examination, the battle is half won because the diagnosis becomes far simpler and treatment follows along. If you cannot and do not think beyond the cornea and conjunctiva, you are certainly going to miss a majority of the conditions which can occur in the horse's eye and in that of every other animal.

Before going on with the conditions of the lids, I want to mention briefly a text that has come out within the last six months. It is Anatomy and Histology of the Eye and Orbit in Domestic Animals by Prince and Diesem. Prince is a research ophthalmologist and Diesem is the anatomist in the Veterinary Division at Ohio State University. They collaborated with others to write this book—an excellent comparative work on the anatomy of the eye and the orbit. It is well written and beautifully illustrated.

Before leaving examination, I might mention that the topical anesthetic agents which we prefer are Ophthaine of Squibb, Opto Drops of Pitman-Moore, or Pontocaine of Winthrop. Opthaine and Opto Drops seem to have the least side-reactions, as far as accompanying edema is concerned, though Pontocaine is satisfactory from the anesthesia standpoint.

[Slide] This is a picture of entropion. Note in this particular case the nictitating membrane is protecting the globe, preventing the lower lid from rubbing on the eyeball. I am going to discuss

this from the standpoint of the surgical procedure that we use in the dog. I have never applied it to the horse, though I do not know why it would not be perfectly feasible.

[Slide] This shows the grasping of the skin below the lower lid and raising a fold in this manner, with the straight hemostat. By allowing that hemostat to remain for 30 seconds or so, that fold of skin remains elevated and then we can determine whether the lid is in proper apposition with the globe and whether the entropion has been corrected or over-corrected before actually removing that portion of skin. If you simply start dissecting a half-circle piece away from the lower lid, often you will under-correct.

[Slide] By this method we avoid that possibility by simply raising this fold and then cutting it off, once we have determined whether we have the proper amount of skin or not. If not, we simply smooth it out with the thumb and grasp a larger or smaller bite, whichever the case may be.

[Slide] This slide shows that portion in the shape of a half moon which has been removed with heavy surgical scissors. The next step is to include a portion of the orbicularis muscle.

[Slide] By grasping the orbicularis muscle at one side of the incision and simply taking a strip out with small straight eye scissors, we are more apt not to have a recurrence of the condition. Especially when there is a great amount of flaccid skin in the area, by removing skin alone, we often have an entropic condition occur again in a few months. Including a portion of the orbicularis muscle will prevent the recurrence of the entropion in practically every instance.

[Slide] This shows the suturing. We use mattress sutures in many instances, although interrupted with a superimposed Stint bandage are satisfactory; an anesthetic ointment is rubbed into the area and in the case of dogs, we use hoods to keep them from getting at the eye.

[Slide] The next slide is of a type of lid neoplasm seen in the dog. I know there are similar lid neoplasms in the horse. In a recent excellent paper in the Fort Dodge Biochemical Review, Vol. 31, No 1 by Dr. Lundvall of Iowa State University, he mentions the removal of lid neoplasms with the use of the hyfrecator or electrocautery equipment of some sort, which we always use in the dog. I presume it certainly could be used in the horse, following a nerve block of that area.

[Slide] This type of neoplasm of the lid (Fig. 5) involves the palpebral conjunctiva and is best removed by the electrocau-

XUM

tery because it minimizes hemorrhage, there is less disfigurement and because one has good, fine control of that procedure, as opposed to dissection with scissors or knife.

[Slide] This shows adenocarcinoma of the upper lid and bulbar and palpebral conjunctiva of the dog, which would appear to be difficult to remove, and it is, as far as I am concerned, by using scissors and knife dissection. With electrocautery at low current and taking it very easy, you can control hemorrhage to the utmost, and actually shave the growth away without recurrence and with far less disfigurement, or the need for plastic surgery to replace the loss of conjunctiva in that area.

[Slide] The next is a melanoma involving the membrana nictitans, which is removed in the same manner.

[Slide] The next condition we are familiar with in man and on down through the species is chemosis or edema of the bulbar conjunctiva, usually the result of an allergic manifestation, such as dust, wind, pollen, and which responds exceedingly well to the antihistamines and corticoids, in combination or individually. When antihistamines were frst introduced, we used them with great success, but the corticoids came along and worked even better. I recall when first going into practice, before the days of corticoids and antihistamines, that we had considerable difficulty with conditions of this sort, and as in man, cases sometimes hang on for a period of several weeks. Drops of adrenalin or preparations of that sort were about all we had to work with. Sub-conjunctival injection of corticoids in this particular area will usually cause a return to normal, sometimes within a few hours, in these particular cases of chemosis.

[Slide] This illustrates the type of injury seen, in man and all animals, in which there is a fluorescein-stained area, indicating a loss of epithelium as a result of going through thickets, woods, so forth. This lesion is very easily missed unless it is stained. An animal may exhibit some photophobia but, upon gross examination of the eye, you will not be able to determine the loss of epithelium in a scratched area. In fact, this particular one was extremely difficult to see, even with magnification, until it was stained, so, I would certainly recommend that you carry fluorescein stain with you at all times. It takes only a moment to stain the eye of the animal, but I would caution you about using the prepared fluorescein solutions in liquid form. It is now pretty well established that Pseudomonas grows well in those particular fluorescein solutions; in fact, research in both human and animal ophthalmology has pointed that out vividly. I know definitely that I lost an eye one time by re-staining with a contaminated fluorescein solution. The drug companies have now come out with

fluorescein-impregnated papers that are obtainable for a cent or so apiece, or you can simply impregnate your own papers, cut them in strips, autoclave them and use in that manner. Most surgical supply houses will be able to supply packages of 100 fluorescein-impregnated papers, which you apply to the lower cul-de-sac, or on which you simply place a few drops of saline and allow the fluorescein to escape from the paper on to the eyeball, giving just the right amount to outline a lesion of this sort, thereby eliminating the possibilities of inoculating an eye with a contaminated fluorescein solution.

Conditions of this sort are simple abrasions. They do not cause too much trouble once they are diagnosed and especially if no greater in exent or depth than this particular case. We simply use an antibiotic preparation for several days. Ordinarily, regeneration takes place within 24 to 48 hours. These are no problem when caught early. They may be missed if you do not use fluorescein, and they may become infected if medication is not used.

[Slide] The next slide shows a fleck of paint or metal (Fig. 6), it could be either, embedded in the upper portion of the cornea, with accompanying edema and vascularization.

There are several ways in which this can be handled under simple tranquilization and topical anesthesia. One is to take the tip of a No. 11 blade and if it is not embedded too deeply, flick it out, or use a tiny curette. Another way is to take an eye dropper, press the ball of the bulb on it and actually suck it off the cornea. It works very nicely, if not too deeply embedded.

[Slide] This slide shows a sliver in the cornea. It is embedded directly in the layers of the cornea. In those instances, of course, it is not possible to simply withdraw the splinter, because it is underneath the epithelial layer and it is necessary to cut down, so to speak, on to the splinter. We have no fear in those particular cases, although it would be necessary, in most instances, in the horse especially, to use general anesthesia, because it is necessary to cut down over the foreign body, whatever it may be — in this case a wood splinter — and then flick it out from underneath the epithelial layer. By cutting down directly over the foreign body, you need have no fear about going through the stroma and into the anterior chamber, because the foreign body lies between the two structures. We use a No. 15 blade, usually, for that purpose.

[Slide] This slide gives the appearance of a granulomatous-type growth in the upper portion of the cornea. That is what we thought it was. We anesthetized this animal and anticipated having to do some sort of dissection and remove that particular area from the cornea. We were not sure whether it was granulation tissue or a neoplasm. On close examination, we noticed a tiny

XUM

foreign body right underneath the epithelium of the growth-like structure. By cutting down through there and withdrawing the splinter, the granulation tissue disappeared of its own accord.

If you see a lesion of that sort in the cornea, you might be suspicious of an embedded foreign body. That is the point I wanted to make in this particular slide.

[Slide] This condition is probably extremely difficult to see; in fact, I cannot make it out here, because of the light, but that is a splinter that goes directly through the stroma into the anterior chamber. Part of the splinter is in the anterior chamber, and the rest is in the stroma of the cornea. Those are difficult to handle; in fact, in one case it was necessary to go through the cornea at the six o'clock position, and actually reach in with forceps and grasp the foreign body from the inside and bring it out, rather than to attempt to cut down through the cornea over the lesion. Foreign bodies can be found in all sizes and shapes and in all parts of the cornea; they can penetrate into the anterior chamber, and they then pose a problem.

[Slide] This is an illustration of hemorrhage underneath the bulbar conjunctiva, in other words, ecchymosis, and also, an intraocular hemorrhage here, right below the pupil, as a result of injury of some sort, usually a blow. As far as hemorrhage under the bulbar conjunctiva is concerned, we do not worry about it at all. We ran an interesting experiment one time on a number of cases when cortisone first came into the picture; making subconjunctival injections of cortisone into one eye of an animal that had bilateral ecchymosis, and allowing the other eye to act as a control. We found that the bulbar conjunctiva would clear in the treated eye in about four days, and about seven days in the case of the control eye. It is just a matter of helping to dissipate the hemorrhage, but this is not important because ordinarily within a week, or at the most ten days, a subconjunctival hemorrhage will dissipate of its own accord.

Blood in the anterior chamber is something different. There are several schools of thought as regards the proper handling of blood in this location. The one I subscribe to, although I some day may change my mind, is that if blood remains in a fluid state and tends to seek a level, so to speak, it should best be left alone; it is not necessary to drain it, because it will drain from the eye of its own accord within a few hours, or by the next day. However, if it is in the form of a clot, especially a massive clot, it is best to invade the eye by paracentesis, attempt to break up the clot and irrigate it, at least the most of it, from the anterior chamber, because a clot that is allowed to remain will tend to organize and proliferate, and often an eye will be lost as a result.

When there is hemorrhage into the anterior chamber, we

use atropine to dilate the pupil so that the pupillary border of the iris will not tend to adhere to the clot.

Another school of thought is of the opinion that one should use a miotic so that the drainage system in the angle of the eye may be opened a little better, and thus allow the clot to be absorbed. However, if a clot has formed, there is very little likelihood of its being absorbed anyway. It is best to draw the pupil away from the clot and atropine is the drug of choice for this purpose.

[Slide] Here is an illustration of an iris prolapse (Fig. 7) that can develop as a result of injury. We have had occasion over the years to be called in consultation on two horses with prolapse of this type, as a result of injury. We have encountered this in a number of cases in the dog, as a result of either injury or ulceration. We know that barbed wire, a nail or ulcer will eventually cause a prolapse, when the lesion extends into the anterior chamber. This iris protrudes into the anterior chamber and through the wound entrance.

[Slide] In this next slide you see what this particular eye looked like one month later (Fig. 8). This is a scar, of course, in this area, but a good adhesion has been achieved between the iris and the cornea. There is very little, if any, loss of vision because the pupillary opening is adequate. Except for the scar, it is not disfigured. You would expect some scarring in that area. The important thing in a prolapse, of course, is to attempt to save the eye. We do that by first snipping off the prolapse flush with the cornea. We then use either one of two methods.

[Slide] This is the first step in the bandaging procedure we have used successfully in the horse, and it is simply a combined roll or pad of any texture, placed over the closed lid. Of course, this is done under general anesthesia. The lids can be closed first by a mattress suture, then this pad is placed over the eye, following the use of atropine and an antibiotic preparation in the eye.

[Slide] Then either Elasticon, which is an elastic-type adhesive, or some similar material, is brought around, starting at the base of the ear—this would be the left ear of this particular horse—the right ear in this area, starting on the base, coming down, catching the medial canthus, missing the commissures of the mouth, and going around two or three times, finally around the front of the ears, and possibly around the neck, to immobilize the eye. A final strip of tape can be used to keep it from slipping. I speak of two cases in the horse in which this worked very well. The aftercare was, of course, up to the referring veterinarian and the owner, but in most instances I remember they cross-tied them but, strangely enough, almost all animals will tolerate a bandage of this sort because it is comfortable. A bandage of this sort is

XUM

allowed to remain for at least three days, and then it can be changed. The purpose of the bandage, of course, is threefold; first, the immobilization that it provides to the eye, which is important following a surgical procedure of this type; second, the warmth it provides, which is conducive to good healing and third, the prevention of infection. So, bandaging can be accomplished successfully in the horse; in fact, it is easier to bandage the horse than it is many of the short-nosed breeds of dogs.

[Slide] This series of slides demonstrates another way of handling prolapses. This method has been used successfully a number of times in our practice in the dog. It has been used successfully in cattle. It possibly could be used in the horse; I don't know why it could not be.

This happens to be a boxer. In this particular instance, we are pre-placing sutures through the membrana nictitans, several millimeters below the superior border. It matters not whether the sutures go completely through the membrana nictitans or through the cartilage, or whether they go merely through the external layer.

The purpose of this particular operation is to bring the membrana nictitans up into the upper fornix and immobilize it by means of sutures, to provide pressure immobilization of the globe. It is an extremely useful procedure in the short-nosed breeds of dogs where bandaging is difficult, if not almost impossible. It has been used successfully in cattle that have had prolapses following ulcerative conditions of the cornea.

[Slide] This slide shows scarification of the inner surface of the membrana nictitans, in the case of a non-healing ulcer. We do this when we are trying to heal an ulcer of the cornea that fails to respond to all other types of medication and handling. In this particular step, the scarification of the inner surface of the third eyelid is not important to the pressure immobilization we are trying to achieve, for instance, in the case of a prolapse. The prolapse is snipped off, wherever it happens to be, and the membrana nictitans brought over in very snug fashion, over the globe to immobilize the latter.

[Slide] This slide shows the position of the sutures as they pass through the upper fornix. This is the upper fornix where the bulbar conjunctiva meets the palpebral conjunctiva of the upper lid. It is through here that we place the double-armed sutures. Sutures have been placed in the medial aspect and lateral aspect of the third eyelid. They will be placed in this direction and in this direction, so we have a taut third eyelid over the globe. This is, in essence, the preparation of a conjunctiva flap, because the third eyelid is conjunctiva, but in this particular case it is

not especially for the purpose of healing an area, as it is for the purpose of protection—immobilizing the globe by pressure.

[Slide] This slide illustrates the position of the buttons. In other words, two sutures in the third eyelid have been placed. Now through the lid and toward the lateral and medial canthus, probably two inches up, the suture is tied over buttons. This suture material happens to be Vetafil. We tie the suture over buttons to anchor the third eyelid up to the upper fornix thus covering the globe, and to prevent pressure necrosis by the use of the buttons.

[Slide] This is the completed picture and shows the sutures in place over the button. The third eyelid is pulled clear up into this area. It is very elastic in the dog, horse and cattle, and can be used in that way There still is an open palpebral fissure through which the eye may be treated by irrigation and the use of atropine and antibiotic preparations several times daily. During the course of the six or seven days following this prolapse operation one does not know what is going on underneath. When the sutures are removed on the sixth or seventh day, there usually is a very good adhesion between the iris and the cornea and from that time on the eye can be treated openly.

I show this particular procedure because I am confident that in cases of prolapse in the horse it could be used successfully as it has been in the cow and dog.

[Slide] Now, corneal disease. The following slides illustrate the type of condition that can result when a prick or abrasion goes undetected and is therefore left untreated or improperly treated. There is deep ulceration and pus in the anterior chamber with accompanying deep vascularization. In these particular conditions, it is usually necessary, or at least best, to drain the anterior chamber by paracentesis. We often cauterize the ulcer but only on its advancing edge, rather than the deep portion, because it is dangerously close to the endothelium, or Descemet's membrane of the cornea. On this particular slide the point I am making again is, that one can prevent many of these eyes from reaching this stage by fluorescein staining, and prescribing properly in the first place — the ulcer or injury will not progress to the deeper structures, and there will not be any deeper involvement of the globe, as often occurs otherwise. Always remember, that in these particular conditions — corticoids are contraindicated; corticoids are excellent preparations when used in practically every condition of the eye, except the acute type of ulceration and similar injuries, because in these latter conditions they tend to delay healing but, more important, they tend to enhance certain infections, especially when the proper antibiotic is not being used. There is no need to use corticoids in ulcers or abrasions of this sort until healing is well under way.

XUM

[Slide] This shows a descemetocele. (Fig. 9) This particular portion of the cornea is clear, as opposed to the remainder. It sometimes gives a feeling of false security because the ulcer has a nice, clear, water-drop appearance and one thinks the ulcer is healing: instead, this is a sign of impending danger because it means that the ulcerative process has proceeded down to Descemet's membrane, the final, or next to the final layer. It happens to be a strong elastic membrane in all species and, for that reason, has not ruptured yet, but it is very near to rupture.

Cases of this sort, whether they occur in the dog, horse, cow or any other species, should be handled with the utmost care because a prolapse is impending. Do not get the idea that this water-drop, clear area is a healing ulcer; the reverse is true. Immediate tranquilization should be instituted so that examination and further treatment can be prescribed. In the dog, we immediately tranquilize or give morphine to effect, to avoid any further handling, and then we proceed to give a general anesthetic, drain the eye of the aqueous by means of paracentesis and use the third eyelid flap, the conjunctival flap, to protect the eye and prevent a prolapse. In most instances, if handled early enough in that manner, prolapse will be prevented. If prolapse develops, it is contained, so that adhesion can take place in that particular area.

[Slide] This slide shows a simple paracentesis (Fig. 10). Paracentesis can be done with a No. 11 Bard-Parker blade, at the six o'clock position. As long as the knife is held parallel to the iris and the stroma of the cornea, one need not be afraid of causing any damage. If, however, it should prick the iris, there would be intraocular hemorrhage, which would defeat the purpose of the operation. If the paracentesis thrust were into the stroma of the cornea quite a scar would be produced and, in addition, one would not be draining the anterior chamber to evacuate any pus or exudate as in a case of iridocyclitis. This is a method we use; some form of traction at the twelve o'clock position, sometimes over on the side. We always do this under general anesthesia, as it would have to be in the horse.

[Slide] This again shows a simple stained ulcer, and I point it out just to tell you our method of handling a simple ulcer, that is — to use a tightly wound cotton applicator (toothpick. preferably) with tincture of iodine. Actually scrub the infected ulcer, so to speak, and rinse with saline, then proceed to use atropine in the eye for the analgesic effect it affords, which, incidentally, is far greater than that afforded by topical anesthetics, especially when the ulcer is of a superficial nature. Then go on to the antibiotic preparation of your choice. Ordinarily, simple ulcerations will respond to simple iodine cautery and antibiotics such as neomycin, bacitracin and chloromycetin.

[Slide] This is granulation tissue of the cornea, an entire area of granulation tissue, showing large blood vessels going into the area. This granulation follows injuries and/or ulceration in which healing has been improper. It has healed but nevertheless become filled in with granulation tissue. It is necessary only to cauterize these vessels and thus destroy the unwanted source of nutrition to the granulation tissue. Corticoids are a valuable adjunct in addition to cauterization of the vessels. We use them by subconjunctival injection. However, in many instances of granulation tissue it is sufficient to simply cauterize these vessels, using electrocautery or heat cautery. This is a simple procedure and the granulation tissue will ordinarily disappear from the scene.

[Slide] This is a case of granulation tissue following ulceration in a cocker's eye. The granulation tissue is building up in this area and also starting over in another area. Again you can see the vessels coming into the granulation tissue. There are three or four major vessels coming into this one.

[Slide] Here is the eye several weeks later. Nothing was done except to cauterize the vessels as they arose in the bulbar conjunctiva. That is all that was necessary. Corticoids may hasten the dissipation of granulation tissue, but the actual cautery is what does the trick.

[Slide] This slide is of superficial vascularization. There are a number of reasons for superficial blood vessel formation in the cornea. One is riboflavin deficiency, though this is not a common condition any more, at least in the dog. Infection or accompanying infection of the conjunctiva, in which vessels invade the area is another cause. Then again, most often we will see superficial blood vessel formation as a result of allergic manifestation, constant irritation, wind, dust, pollens and so forth. These respond exceedingly well to the use of corticoids in one form or another, but by far the best method is the subconjunctival injection of prednisolone. One can "melt" superficial blood vessels in a matter of a few days; at least by the fifth day when the third injection is made, most of the vascularization has disappeared from the scene.

[Slide] This slide will illustrate the method of subconjunctival injection (Fig. 11). In the horse, since there is continuity between the palpebral and bulbar conjunctiva, it makes little difference whether you use palpebral or bulbar conjunctiva for the injection. Honestly, I think that subconjunctival injections of prednisolone are not being used enough in this country. We are using it in vascularization processes, granulation tissue and certain disease processes involving the dog's eye. I have in mind the German Shepherd with a peculiar disease, in which prednisolone at 48-hour intervals will simply melt the lesions from the cornea. In the horse I would think you would use about 1 cc or 20 mg of

XUM

hydeltrasol, Merck's Hydeltra-T.B.A. We are using that almost exclusively in the dog. It is one of the more concentrated prednisolones. You only need ¼ cc in the dog, or 5 mg. Under adequate topical anesthesia and perhaps, additional tranquilization, this can be readily achieved. It must be repeated in 48 hours, however, because the effect starts to work off. Although you will note a residue for many weeks, and sometimes even months afterwards, it does not mean that this is corticoid exerting its effect; it is simply solution residue. We use a 25 gauge needle in the dog, and twist the needle as we remove it from the conjunctiva, thereby preventing leakage.

Finally, I would like to suggest several first-aid preparations that you might recommend to the owner of a horse over the telephone, or have him use around the stable.

One is an excellent soothing and antiseptic wash for any animal; in fact, I carry a bottle of this with me on all hunting trips and when driving long distances, to soothe my own eyes. It is a preparation that can be made very easily at the farm or the stable, by taking a moderately heaping teaspoon each of bicarbonate of soda, borax and table salt, and dissolving these in a quart of boiled water. They readily dissolve with the exception of the borax, some of which settles to the bottom. To this preparation, add a tablespoon of glycerin. You can use either the supernatant fluid or the filtered preparation for irrigation of discharges from the eye, foreign bodies, weed seeds and so on, without injury to the eye. It is a very soothing preparation. I abhor the use of plain tap water. Tap water itself is irritating to the eye, and every animal resents it, whereas this particular preparation is soothing. Here is one you can recommend over the phone and have them keep around to use at anytime. Especially when there is a copious discharge, it is almost useless to use antibiotic preparations in solution or ointment form, unless the discharges are first cleaned from the eye. We prefer this type of preparation in any animal, for the cleaning of discharges before the use of an antibiotic preparation.

In addition, as a first-aid treatment, recommend either castor oil or cod liver oil, preferably castor oil. It is absolutely non-irritating. It affords a great amount of relief following abrasions and injuries of that sort, until your services can be obtained.

I have mentioned the contraindications of the corticoids. I want to re-emphasize, in the case of an acute ulceration or abrasion, that only preparations without corticoids should be used, at least for the first few days, until the condition is under control or until healing is well in evidence; then they can be used safely. There are no other contraindications for the use of the corticoids. We are extremely pleased that they have come along and have been of such valuable help in ophthalmology. They are not helpful in certain

degenerative conditions and that sort of thing, but they are not contraindicated in eyes except in cases of acute ulceration and abrasions.

CHAIRMAN REED: What is the most common cause of atrophy of the eyeball following descemetocele?

DR. MAGRANE: Following a descemetocele? You mean there has actually been a prolapse?

CHAIRMAN REED: Yes.

DR. MAGRANE: That is an interesting question, because this atrophy may follow the removal of a subluxated lens in the dog's eye. Just as an example, you can bathe the eye and remove the lens in ten cases of secondary glaucoma in the wire-haired terrier, and nine out of ten will be perfectly all right, but in the tenth, the eye will start shrinking after a few weeks or few months and proceed to phthisis bulbi. This also applies to cases of prolapse. It is a condition that can follow repeated attacks of periodic ophthalmia. I don't have a good explanation for its occurrence: in fact, I have never read an explanation. In human medicine they can operate on bilateral cataract and have one shrink and the other remain perfectly normal, as far as contour and tension etc. is concerned. I am sorry I cannot give you a specific reason.

DR. CARRICABURU: When you are aspirating the eye, using general anesthesia in the horse, how do you control nystagmus?

DR. MAGRANE: When I was in graduate work at Pennsylvania, Raker and Jenny put some horses down for me. We did experimental eye surgery. They had to be very deep in order to prevent nystagmus. They got them down to the point where they were very well anesthetized! I worried, even though they didn't.

In addition to general anesthesia, there is no reason why nerve blocks cannot be performed. There is what is known as the O'Brien block, used in the human and in the dog. I should think it could be used in the horse. It is a blocking of the extra-ocular muscles; in fact you can block the recti muscles directly in the horse and thus control nystagmus, and so prevent putting them down so deeply. It is a matter of working out mechanics and blocks. It should be a valuable adjunct to general anesthesia in the equine.

QUESTION: Is the tincture of iodine strong or weak?

DR. MAGRANE: It makes no difference, we have used both.

QUESTION: Sometimes it is difficult for me at least, to judge the turning point or healing in these ulcers.

DR. MAGRANE: The question is, how do you judge the healing time? It is difficult, I will admit, for everyone. Fluorescein

XUM

again plays an important part here. It is not only valuable from the standpoint of diagnosis but also prognosis. For instance, if you re-examine an eye in 48 hours, it is well to use fluorescein strips again, to determine the extent of healing. It is difficult to see how much healing is taking place without adequate staining. We often are surprised at the amount of healing that has taken place. You may look at the eye and there will be some opacity in the area of healing. The eye may not look so well, yet be coming along nicely, whereas the reverse is true on the one slide I showed of descemetocele. The ulcer appears to be healing, but the reverse is taking place. Fluorescein is valuable in that, by using it repeatedly, you can determine whether healing is taking place toward the center and also whether the crater is filling in.

QUESTION: In your experience is there a difference between the horse's eye and the dog's, in that in the horse, the corneal ulcer heals and in the dog you have a lot of trouble?

DR. MAGRANE: I really can't answer from the standpoint of ulcers in the horse, because I am so worried about the ulcer that won't heal in the dog's eye. I don't have any good explanation for this, other than to say that you are fortunate that this takes place, because a dog's eye is so much easier to treat. Maybe somebody else will differ with you. In certain breeds, like Pekingese and in certain strains of other breeds, the corneas are subjected to the most virulent type of ulcerations. No matter what you use, they go on to prolapse. Whether that is an experience with certain types of horses or not, I do not know. It certainly runs in breeds; whereas certain breeds can have an ulcer which heals in a matter of 24 or 48 hours, many prolapse in that length of time.

QUESTION: When pus is present in the anterior chamber and paracentesis is carried out, does the pus run out easily?

DR. MAGRANE: I was going to spend some time on that problem tomorrow, but I will mention it now. Pus clots are sometimes quite well organized and it is necessary to reach in with what we call a dull iris hook or eye tissue forceps and actually grasp the clot and bring it up to and tease it through the opening. In some instances it is necessary to irrigate; in others it is impossible to disorganize a clot. This is not as important as the fact that you have drained the anterior chamber. Paracentesis is for the purpose of (a) mechanically removing exudates from the anterior chamber and (b) draining the stagnant aqueous which is replaced by secondary or plasmoid aqueous, which is extremely high in antibody titer as compared to the original and thereby is beneficial to whatever the disease process happens to be, whether an ulcer or periodic ophthalmia. This is the more important of the two reasons. The mechanical removal is secondary. If it can be achieved, fine. We do not mess around inside the eye too long trying to get the pus drained.

QUESTION: You described using the membrana nictitans as a conjunctival flap and that you suture it. Will you describe your aftercare when you take the sutures out?

DR. MAGRANE: The aftercare consists of the irrigation of any secretions through the still open palpebral fissure, though you cannot see the globe. Whatever the case happens to be, if it were a prolapse for instance, we would use atropine in that eye and we would use an antibiotic preparation several times a day. This would usually be continued for several days following removal of the sutures.

In addition to the use of a third eyelid flap, I would recommend bandaging for the protection it affords; the dressing to be changed at the end of two or three days. It is not necessary to change dressings earlier.

QUESTION: Does an adhesion form between the flap and the cornea?

DR. MAGRANE: The most it will stay there is a few seconds and then drop down. Even when one scarifies and the ulcer bleeds profusely, it will not adhere.

QUESTION: Is it generally necessary to cauterize these corneal ulcers more than once?

DR. MAGRANE: In some instances you may cauterize more than once, but repeated cauterization is certainly not indicated. Some men have the opinion that after 24 or 48 hours they do not see much change and that they should cauterize again. They are not giving it a chance to heal. Let's put it this way: there has been more overtreatment of ulcer than there has been undertreatment. This applies also to the use of antibiotics and other preparations, in ointment or liquid form. Essentially, the thing to remember about corneal ulcers is first, to get the infection under control; cauterization is one way, and antibiotic administration is the second. When infection is under control and healing is under way, get the blazes out of there with your preparations, because anything you put in the eye is apt to, in fact most of the time will interfere with regeneration of corneal epithelium. So, it is best to get an infection under control; once it starts to heal, leave everything out.

This brings to light something that bothers me. I know that it is very popular, especially in large animal practice, to use powders of one sort or other, in the eye. I know in range cattle it is about all you can do. I certainly would prefer that it be in liquid form rather than powder. The powder is definitely abrasive to the eye. If an ulcerative process was present, the effect is like throwing sand into the eye. For horses I would prefer a solution of a preparation rather than a powder for topical use in these eyes.

XUM

QUESTION: Do you have any opinions on the use of radiation agents for corneal granulation?

DR. MAGRANE: We have had a beta ray applicator for a number of years; we used x-ray before that. The corticoids have pretty much replaced the need for irradiation. It takes far longer for beta ray to destroy granulation tissue or blood vessels than by using simple heat perimitry or even subconjunctival corticoids. We use beta ray on occasion as an adjunct but not to nearly the extent as formerly. I do not think it is a necessary piece of equipment to have around.

QUESTION: Do you always invade the anterior chamber on the ventral aspect?

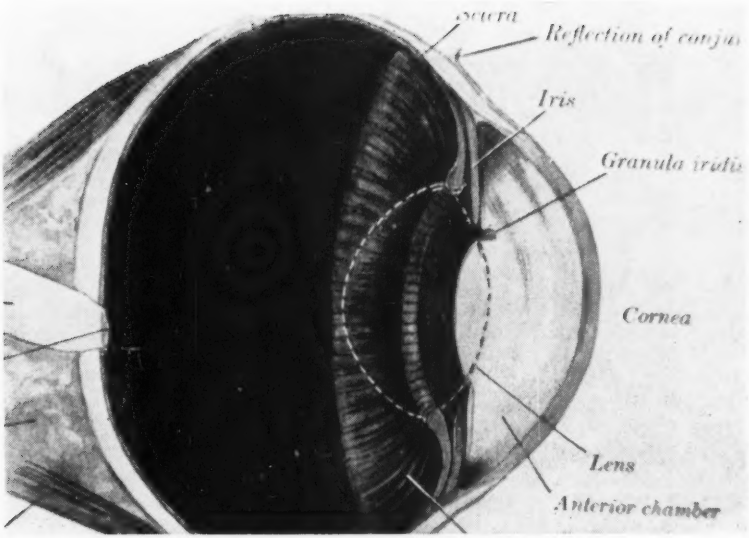
DR. MAGRANE: Yes. The reason is from the gravity standpoint. Drainage is far better, from the mechanical standpoint, from the ventral position. With the use of an eye muscle hook, one can get in underneath the membrana nictitans on the ventral aspect of the globe, bring it right forward and with a lid retractor reach the 6 o'clock posiion. It is a matter of using certain pieces of equipment. I realize it is difficult to hit the 6 o'clock position, at least near the sclera, but somewhere in the ventral position will do; it need not be right at 6.

QUESTION: In our clinic we have seen what appeared to us to be viral warts invading the eye of the dog. Have you seen these?

DR. MAGRANE: Yes. I have had two, one on the third eyelid, the other in the bulbar conjunctiva at 2 o'clock. So you haven't seen anything that is brand new, though I would say it is rare. They will disappear or they can be snipped off.

DR. C. H. REID: How about the use of systemic antibiotics in conjunction with local administration?

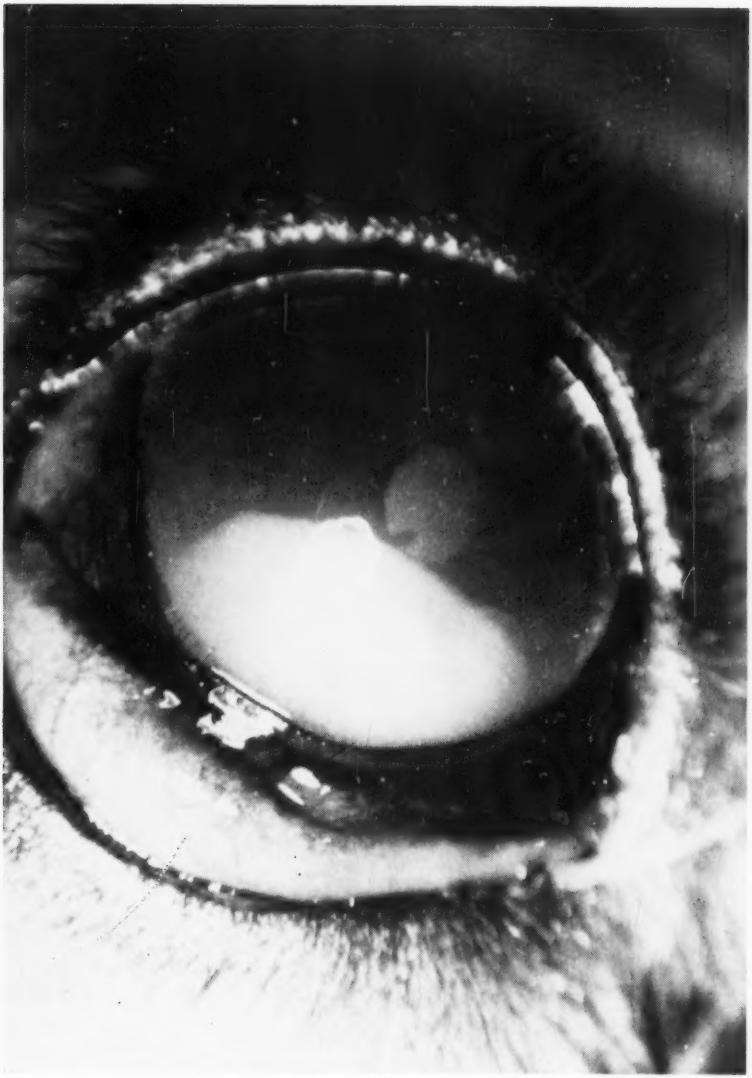
DR. MAGRANE: If there is deep ulceration and a badly infected eye and you think you are going to do any good with penicillin from a systemic standpoint, stop using it. About the only antibiotic that does any good, as far as reaching the interior of the eye, is chloromycetin; this applies to the sulfonamides, too. Massive doses of penicillin, streptomycin, will not penetrate to the required depth. When dealing with external lesions, topical application is sufficient. When there is a break in the epithelium, those topical preparations will penetrate through the stroma of the cornea.



MAGRANE; Fig. 1. Equine eye — sagittal section.

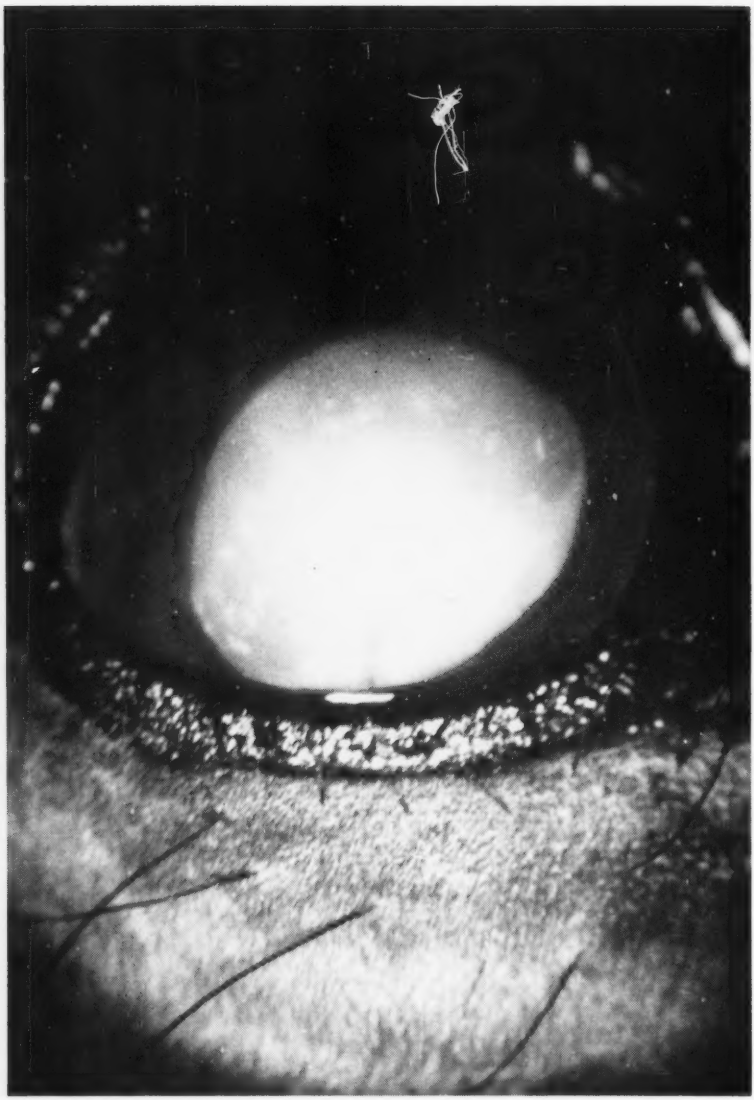
XUM

MAGRANE; Fig. 2. Normal equine eye.



MAGRANE; Fig. 3. Hypopyon, canine.

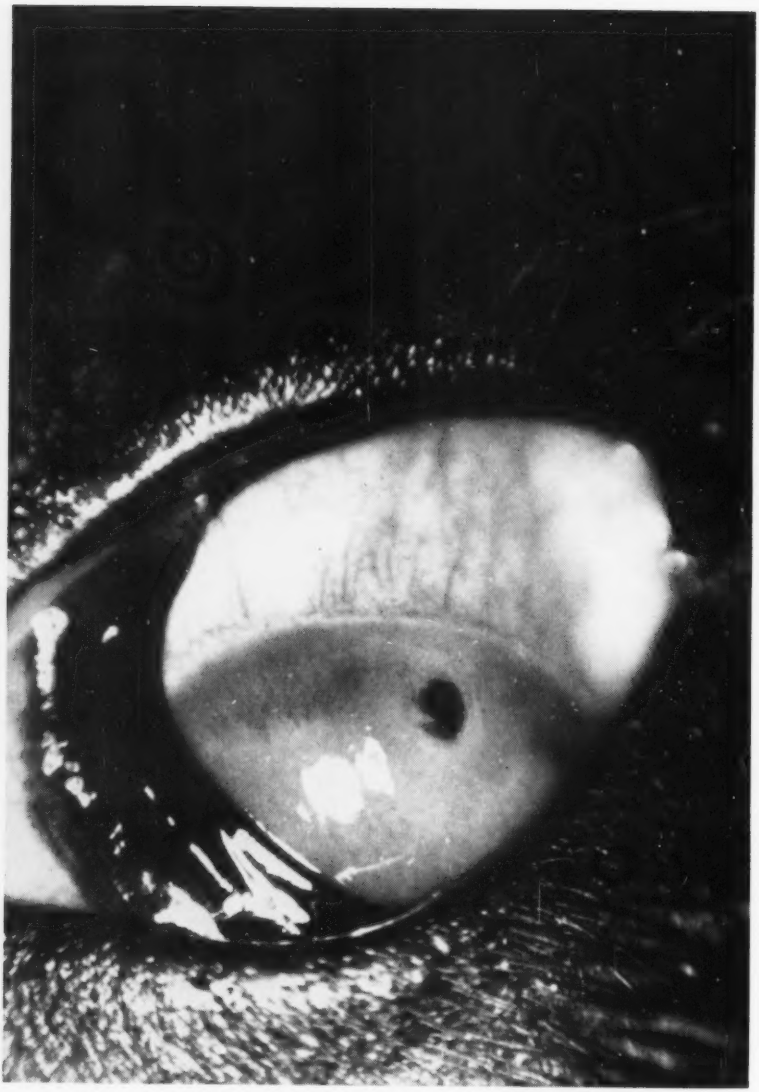
XUM



MAGRANE; Fig. 4. Luxated cataractous lens in anterior chamber. A sequel to periodic ophthalmia.

MAGRANE; Fig. 5. Neoplasm, upper lid, canine.

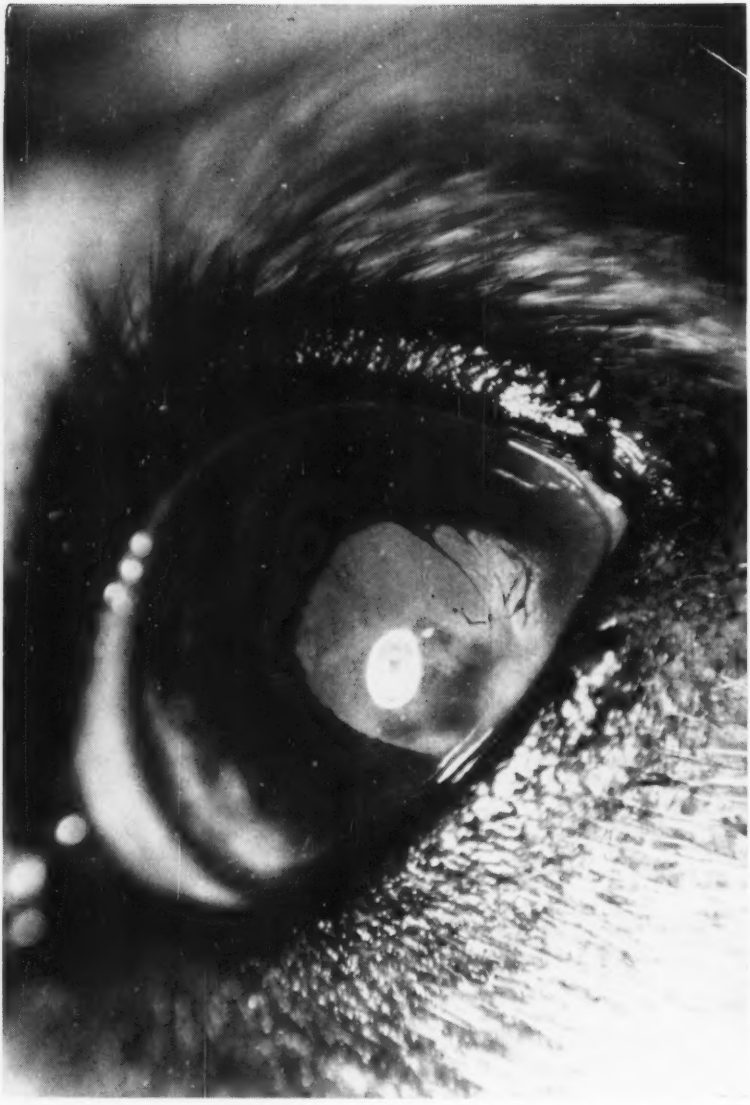
XUM



MAGRANE; Fig. 6. Foreign body embedded in cornea, canine.

MAGRANE; Fig. 7. Prolapsed iris, canine.

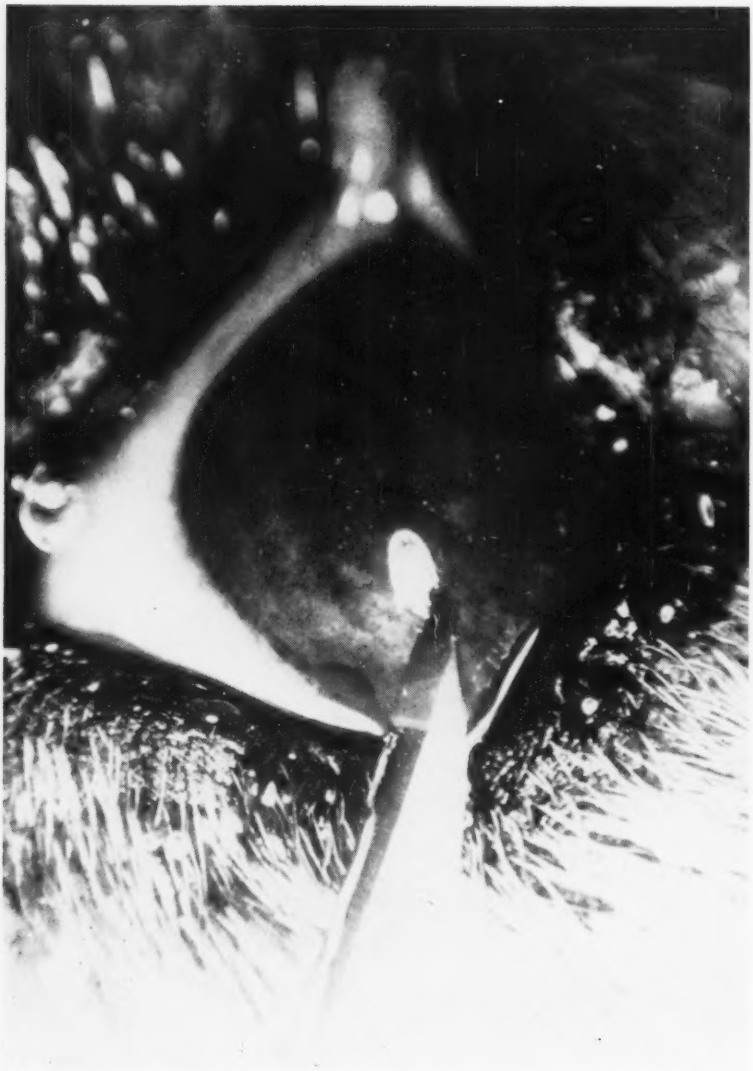
XUM



MAGRANE; Fig. 8. The healed eye following correction of prolapse, canine.

MAGRANE; Fig. 9. Descemetocele, canine.

XUM



MAGRANE; Fig. 10. Paracentesis of anterior chamber, canine.

MAGRANE; Fig. 11. Subconjunctival injection.

XUM

SNAKEBITE POISONING IN HORSES

C. H. Burger

INTRODUCTION:

The treatment of bites by poisonous snakes has been a subject for discussion by some of the most learned men of their time, including Galen and Pliny, nearly two thousand years ago. Even as recently as 1888, Yarrow, a surgeon in the United States Army, treated snakebites by thrusting the bitten extremity into a freshly killed deer or cow. This, of course, must now be relegated to the level of not-so-harmless folk-lore[9].

In San Antonio, Texas, in 1926, Jackson, a physician, in collaboration with Rhea, a veterinarian, tested hundreds of remedies on bitten dogs and found all of them to be worthless. However, their next approach to the problem was the removal of the venom by incision and suction. This method reduced mortality in a large series of human cases from 15 per cent to about 2 per cent[8].

It is estimated that several hundred horses are bitten annually in the United States by poisonous snakes. Estimates of mortality range from 10 to 20 per cent. Morbidity is 100 per cent but can be greatly reduced by proper treatment. These estimates are based on a recent Florida survey among practicing veterinarians[1], personal experience of the author and his colleagues and a current poll of equine practitioners.

GEOGRAPHIC DISTRIBUTION:

Poisonous snakes are found in 48 states. Because of its size and aggressiveness, the most dangerous venomous snake in the United States is the large rattlesnake (genus Crotalus). The venom of the pit viper (rattlesnake, copperhead and moccasin) is mainly hemotoxic and proteolytic, causing extreme local swelling and a very rapid but weakening heart beat[2]. The venom of the coral snake is essentially a neuro-toxin and is not considered a major source of poisoning in the horse due to the less aggressive attitude and limited geographic distribution of this snake.

SEASONAL INCIDENCE:

Most bites are inflicted during the first warm days of spring, but cases may occur throughout the summer. Occasionally, a horse is bitten during the winter months if the temperature rises sufficiently to warm the snakes and make them active. Courtwright, an Arizona practitioner, can recall three such cases over a twenty year period[3].

XUM

CLINICAL SIGNS:

Horses are most frequently bitten on the nose and about the head, less frequently on the legs and chest. Bites on the nose and head are by far the most serious because of the tremendous swelling which rapidly follows a bite. Within a few minutes, the nostrils become edematous, the nasal mucosa swells and later, blood-stained, frothy exudate may hang from each nostril. The eyelids are swollen to such an extent that ptosis is obvious. The ears become edematous and are directed laterally. Severe dyspnea develops with accompanying stenotic respiratory sounds which are audible at a great distance.

The extreme depression and feeling of helplessness makes even unbroken horses amenable to treatmnt.

TREATMENT:

The most important emergency measure for a horse showing respiratory distress is tracheotomy for which the administration of a local anesthetic is unnecessary. The treatment of leg bites should be directed to the localization of the venom by means of a tourniquet judiciously applied for a 20 minute period, then released for 2 or 3 minutes and re-applied proximally. The total time of tourniquet pressure varies from 2 to 3 hours. Antivenin may be injected at the site of the bite. Incision and suction of the fang wounds are useless unless performed within a few minutes of the infliction of the bite[4].

Ice packs and cold water help to slow the spread of the venom, and reduce the swelling and pain and often are the only available first aid measures.

Therapy must include some of the following agents, depending on the lapse of time following the bite and the economic value of the horse. We refer to them as the four A's of snakebite therapy.

1. ANTI-INFLAMMATORY HORMONES:

Cortisone, its related analogues and A.C.T.H. are most useful in reducing morbidity and mortality in the horse. This form of treatment was reported effective in the horse in 1956[5] following a report of its value in humans by Hoback and Green in 1953[6].

With cortisone therapy, the signs will subside and become milder and there will be less tissue necrosis and sloughing.

2. ANTIBIOTICS:

Penicillin and streptomycin combinations or broad-spectrum antibiotics are definitely indicated because the bacterial flora in snakes' mouths has been shown to contain coliform and enteric bacteria[7] and the necrotic tissue is an ideal medium for many saprophytes.

3. ANTITOXIN:

The organisms of tetanus and gas gangrene have been isolated from the venom of a large series of snakes hence the rationale for the use of agents to combat their effect.

4. ANTIVENIN:

Specific antivenin therapy, including injections into the site of the bite and parenterally is costly and its value greatly diminished in all but the very early case. The risk of anaphylactic reaction must be kept in mind since the product is a serum obtained by the hyper-immunization of horses with a mixture of snake venoms.

SUPPORTIVE THERAPY:

The administration is indicated of saline, dextrose, blood (blood substitutes) and vitamins to combat shock and dehydration and to furnish nutrition.

Nor-epinephrine is effective in cases of shock and circulatory collapse. Calcium gluconate may be useful in reducing hemolysis caused by the action of the venom.

The parenteral use of proteolytic enzymes to reduce swelling is probably indicated.

If the case is seen within a few minutes of infliction of the bite, surgical measures include cruciate incisions, one-quarter of an inch deep, over the fang marks and the application of suction to the area. As already mentioned, tracheotomy may be a life-saving procedure if dyspnea is severe.

Physical measures include the use of the previously mentioned tourniquet, ice packs, cold water and also ethyl chloride spray. Cryotherapy if prolonged beyond 2 or 3 hours, may lead to tissue destruction as a result of freezing of the part.

CONTRA-INDICATIONS:

(1) Experimentally, anti-histamines have been shown to exert a deleterious effect on cases of snake bite in mice and dogs[1]. This has not yet been substantiated in the horse but it is a well-considered opinion that they are contra-indicated in snake bite in the horse.

(2) The excessive application of cryotherapy as previously stated and

(3) The indiscriminate use of a tourniquet are also contra-indicated.

SEQUELAE:

The recovery from the effects of a snake bite can be a long, drawn-out affair, associated with necrotic sloughs and great loss of bodily condition.

XUM

DIFFERENTIAL DIAGNOSIS:

Fractures about the head may pose a problem especially during warm weather in known, snake-infested areas. The differentiation may not be made for several days, until swelling has subsided and corroborative evidence such as a bone chip or loose tooth is noted on clinical or radiographic examination.

Infectious diseases, particularly the strangles-influenza complex may be confused with snake bite but the presence of fever and purulent exudates helps in deciding that an infectious process is present.

Purpura hemorrhagica may be differentiated on the basis of widespread mucosal hemorrhages along with extreme edema of the legs and ventral part of the abdomen.

The stings of bees, wasps and other poisonous insects may offer difficulty since the effects may differ only in the degree of tissue reaction.

PREVENTION:

Rattlesnakes can be reduced in number by the annual systematic poisoning of squirrels and other rodents and by grazing pigs on the pastures and fields where snakes are found.

DISCUSSION:

Several factors influence the severity of the effects of snakebite in the horse. These include the size and type of snake, the weight of the victim in relation to the amount of venom deposited, the site of the bite and condition of the horse. Bites on the face, as already mentioned, are most dangerous. Most untreated horses will not die from snakebite but the period of morbidity is a major consideration.

Most practitioners who have had considerable experience of treating snake bite, believe that the period of time elapsing between infliction of the bite and institution of treatment is the most important factor in reducing mortality and morbidity. The breed of the horse has a bearing on the severity of the effects of the bite, cold-blooded horses being more refractory than Thoroughbreds or Arabians.

If possible, the veterinarian should go to the patient instead of having it ridden or trucked to the office, since the movement and exertion lessen the chances of a successful outcome.

REFERENCES

1. PARRISH, Henry M., SCATTERDAY, James E.: "A Survey of Poisonous Snakebites Among Domestic Animals in Florida." Vet. Med. 52: (1957), 135.

2. SMITH, H. A. and JONES, T. C.: **Veterinary Pathology,** Lea and Febiger, Philadelphia, (1957), 535.
3. COURTWRIGHT, E. L.: Personal Communication, (1961).
4. AHUJA, M. L.: **Snakebite, Current Therapy,** W. B. Saunders Co., Philadelphia and London, (1960), 719.
5. BURGER, Charles H.: "Special Problems in Pet Horse Practice." Presented to the Calif. State Veterinary Medical Association Midwinter Conference, (1956).
6. HOBACK, W. W. and GREEN, T. W.: "Treatment of Snake Venom Poisoning with Cortisone and Corticotropin, J.A.M.A. 152: (1953), 236.
7. PARRISH, H. M., McLAURIN, A., and TUTTLE, R. L.: "North American Pit Vipers: Bacterial Flora of the Mouths, and Venom Glands." Virginia Med. Monthly, 83: (1956), 383.
8. JACKSON, D.: "Rattlesnake Bite: Its Treatment and Care." Translated from Spanish by Stone, L. E. in Madera County Health Office Bulletin, June, 1936.
9. RUSSELL, F. E.: "Folk-lore Remedies Suggested for Tigersnake Bite." Western Med., March, 1961.

QUESTION: Dr. Burger, you must have been studying during one of the former presentations in which the speaker mentioned that one could apply a tourniquet for two and one-half hours of steady pressure. Want to comment on this as far as the horse is concerned, in releasing it every twenty minutes?

DR. BURGER: You are not quoting him correctly. He said he was using pressure tourniquet. My understanding was that it was equalized all the way around, not some old piece of rubber tubing. I will stand by my statement that tourniquets probably cause more harm than good, if they are not used properly.

DR. C. H. REID: Ever use injection of camphor and oil to support the circulation?

DR. BURGER: No. I think it might be indicated as is norepinephrine in shock. Thank you for bringing up that point.

DR. KESTER: What was the question?

DR. BURGER: Ever use a stimulant such as camphorated oil?

DR. REID: Camphor and oil, 25 per cent solution of camphor in cottonseed oil.

DR. BURGER: The cottonseed oil would scare me a little bit. One other factor that affects the severity of the bite episode is the anger of the snake. Apparently, a snake that is very mad will really let go with a lot of venom, as opposed to one that gives a light blow and isn't too angry about the whole thing. With snakes in captivity, apparently the venom is not as bad as the wild fellow living in the field. I think if you keep snakes in captivity, your results from venom studies are not quite as valid as those in the field. I don't think half the people who work with snakes consider this point.

XUM

QUESTION: How are most of the toxins attacked by the body? How does detoxification occur?

DR. BURGER: The question is, how does the body detoxify the toxins involved? Apparently, to a great extent, it doesn't, and that is why we have death and tremendous swelling. I think the body tends to try to combat it by diluting with great edema. This is a gross pathological observation, without scientific backing.

DR. T. E. DUNKIN: Last year, Dr. Carroll from Pueblo, Colorado, brought out the fact of inserting plastic tubing in the nostril when the snake bite involved the head region. Have you used this?

DR. BURGER: No, but I have used tracheotomy. It has been a life-saving procedure. There is one gentleman from Arizona who has seen a considerable number of cases of snake bite, and he suggests on these nose bites, if there is a great deal of edema around the nose, taking a little aluminum metal band and propping open the nostril; this obviates the necessity for tracheotomy in certain cases when the swelling is towards the tip of the nose.

QUESTION: What about the use of 10 per cent calcium gluconate injected locally around the tissues?

DR. BURGER: I have not heard of it injected locally. It has been recommended parenterally, in the belief that it cuts down hemolysis. There is no real good experimental evidence that shows it does this, but many people think that it does, and that it is indicated in a snake bite.

DR. DUNKIN: What is the solution?

DR. BURGER: Calcium gluconate solution injected locally around the bite. There are many people who think it helps to inject calcium gluconate parenterally.

QUESTION: Is adrenosem salicylate(R)* of any value?

DR. BURGER: Have not used it.

QUESTION: I was just wondering after how much time following a bite you think a tourniquet is of no more value.

DR. BURGER: How much time after the bite is a tourniquet no longer of value? When there is considerable swelling that has climbed up the leg, I would say that the tourniquet is of less value after an hour or so, but it would depend on the amount of swelling and the area bitten.

I know that many ranches have cortisone in the ice-box and, as soon as they get a case of snake bite, they inject cortisone and give me a call. It has worked so well, that for the last year we have not been able to get movies of a horse really in distress.

---

*Adrenosem Salicylate (brand of Carbazochrome Salicylate) is a product of S. E. Massengill Co., Bristol, Tenn.

CLINICO-PATHOLOGIC CONFERENCE

Moderator: C. W. RAKER

Panel: S. P. DEY

E. DILLMAN

F. J. MILNE

CHAIRMAN REED: This morning, we are to introduce something new to our annual meeting — a Clinico-pathologic Conference. I have had the opportunity of taking a couple of postgraduate medical courses in New York, and there they do this same thing. They hand out a sheet and on it are several case histories. From this you have an opportunity to study the cases and make a diagnosis.

Dr. Charlie Raker is going to handle this session. He intends to show a movie or slides of the case and have someone discuss the various possibilities, and then you form your own conclusion as to the diagnosis.

The object of this is to stimulate one's own thinking and ability to make a diagnosis.

DR. RAKER: Thank you, Dr. Reed. As Dr. Reed pointed out, we hold these conferences routinely at the University of Pennsylvania School of Veterinary Medicine and I know that many other veterinary schools, associations and local groups are doing likewise. It is quite stimulating and usually puts the discusser in quite a spot because he does not know the diagnosis. Because of this, sometimes these sessions become rather humorous and sometimes, for the discusser, rather embarrassing, but this is the way we learn.

There are two cases for discussion. I will give a brief background on the first case. Dr. William Wright originally had the case. Dr. Dillman was called in to represent the insurance company, and Dr. Jenny also was called in as a consultant. I have never seen the case. I am going to show a short motion picture and Dr. Dey, who is one of my associates at Pennsylvania, is going to discuss it. He has not seen the case nor does he know the diagnosis. I do know the diagnosis because I have the necropsy report and the pathologist's report, which I will give at the end.

Now, let us just quickly review the case. Unfortunately, in this case there is not a lot of material available. Dr. Dillman has seen it and I have asked him to come up here because there may be some questions you would want to ask after the discusser gives his comments.

XUM

This is a four-year-old thoroughbred stud with lameness in the left hind leg. The racing record has been reproduced here from June 7 to July 8. During this period of about a month, the horse had a total of six starts at intervals of ten, four, seven, six and four days. They were all stakes races, and they were all at a distance of one and one eighth to one and one half miles. This, I think we all agree, is pushing a good stake horse rather hard.

The horse was slightly lame after the last race on July 8. He was taken out of training. The condition was diagnosed as a neuromuscular disorder and the owner was advised to have the horse turned out in a small paddock. In the ensuing three weeks, the stud showed increased atrophy of the left hind leg and by the middle of August, had difficulty getting up by himself. At the time of Dr. Jenny's examination on August 24, the stud could not get up without help. He exhibited considerable muscle atrophy, especially of the gluteal and quadriceps region.

[Slides] These show the appearance of the horse on August 24, and the severe atrophy of the gluteal and quadriceps muscle groups.

[Motion picture] There was extreme lameness of the left hind leg. The fetlock was held in flexion and only occasionally would the stud put the left hind foot down correctly. Most times he would knuckle over and drag the toe. The patella did luxate laterally, but was replaced spontaneously as the patient stretched his hind leg out posteriorly. There seemed to be a slightly increased reaction to pin pricks over the lateral aspect of the thigh. There was no deviation in the carriage of the tail, nor was the tail movement inhibited. There was good tonus of the sphincter ani and there were no complications with urination. Rectal examination on the standing and moving horse was negative.

This horse became progressively worse from July 8 until August 26, when euthanasia was carried out.

I am now going to turn it over to Dr. Dey and let him proceed to discuss the case, after which I shall throw it open for discussion and diagnosis. Then, in conclusion, I shall give a summary of the pathologist's report and show some photomicrographs of the affected tissues.

DR. DEY: In reviewing the history and progress of this horse, it appears that the most prominent clinical signs are a marked degree of unilateral atrophy in the gluteal and quadriceps groups of muscles and a paresis of the left hind leg.

Atrophy of a muscle may be caused by several conditions, but three which immediately come to mind pertaining to this case are:

1. Disuse atrophy which is seen in a leg due to a pathological lesion at some point, altering the normal use of the limb.

2. A direct insult to a muscle resulting in hemorrhage, necrosis and acute atrophy of the involved muscle.
3. Injury to the vascular or nerve supply to a muscle resulting in acute atrophy.

Disuse atrophy is an insidious process developing over a variable period of time, depending on the severity of the pathological lesion causing the lameness. This may result in only a slight deviation in muscular symmetry over the croup unless associated with marked pain and limitation of function. However, in this case we do not have a history indicating that the hind leg problem has been developing over a long period of time and this, of course, would tend to rule out disuse atrophy. Secondly, in this type of case one would not expect an increase in sensation to pin pricking over the lateral side of the thigh.

There is no history of direct insult to these muscle groups nor is there a history of falling or being cast in the stall. Furthermore, physical examination did not reveal a hematoma, abrasion of the skin, etc., all of which would tend to rule out direct trauma.

The problem then would appear to be either neural or vascular in origin.

A rectal examination was negative for the presence of a thrombus in the anterior and posterior iliac arteries as well as the terminal aorta. There was no detectable difference in the temperature of the extremities of the hind leg and the pulse taken at the great metatarsal arteries on both the left and right hind legs was apparently of the same quality — all of which would tend to rule out a vascular problem. It might also be added at this point that the quadriceps and gluteal groups of muscles do not have the same arterial supply.

This leaves us with a neural problem as the probable cause of the present condition. In referring to neural disorders, one must consider the categories into which these problems can fall. Graphically, neural problems can be broken down into two categories: 1. central and 2. peripheral.

Problems involving the brain such as tumors or systemic infections as a rule affect the body as a whole and not just one portion. Furthermore, we find here a horse with a bright, healthy appearance, good appetite, with normal bowel movements and urination. These findings also would tend to rule out brain involvement. If an inflammatory lesion of the brain were present, I would expect to see a decrease in alertness even to the extent of stupor or marked depression or perhaps an animal exhibiting hyperexcitability.

Going now to the spinal cord, we find that examination of the neck did not reveal any asymmetry of structure nor did movement

XUM

of the head in various positions seem to affect the colt in any manner. In wobbles, asymmetry of the croup muscles is not one of the signs of this disease and furthermore in this condition as a rule both hind legs are equally affected.

Disease of the spinal cord in the lumbar region cannot possibly be ruled out and must remain in the picture. However, the literature indicates that these problems are again bilateral, in which case one would expect to see bilateral changes in muscle atrophy, sensitivity, the same temperature over both legs, be it abnormal or otherwise, etc. Secondly, in these cases there is usually an ataxia of both hind legs. Bone or joint disease in this area has been ruled out to some extent by the rectal examination. The scoliosis I believe can be explained by the horse placing most of his weight on the right hind leg and therefore being forced to bring it toward the center of the body to form a tripod for better support. This produced a convexity of the spinal column to the right side.

It would appear then that the principal problem involves the peripheral nervous system, in particular the lumbar nerves as they leave the spinal cord.

This colt, therefore, in all probability had a peripheral neuritis (inflammation of a nerve due to infection, trauma, pressure, chemical or toxic substance, etc.). A tumor or abscess in this case would be the two most likely disease processes to bring pressure to bear on the nerves. A less likely cause would be bone disease with new bone formation. It would be my opinion that the nerves involved in this case include the femoral and probably gluteal. I say this because involvement of the extensor muscles appears greater than is the case with the flexor muscles. I am unable to make a final diagnosis in this case; however, in my opinion, a peripheral neuritis due to a tumor or abscess resulting in pressure on the peripheral nerves in the lumbar region of the cord is the most likely diagnosis. These two entities might possibly have been differentiated by additional clinical procedures and laboratory studies.

DR. RAKER: You have heard Dr. Dey's discussion of this case. His final diagnosis is basically a peripheral neuritis. Is that correct, Dr. Dey?

DR. DEY: Yes.

DR. RAKER: The etiology of which, at the moment, is not determined. You have ruled out involvement of the central nervous system and spinal cord?

DR. DEY: Yes.

DR. RAKER: Now I would like to have some discussion, comments or questions. This is why it was put on the program. So, let's have it.

QUESTION: How good was this horse in the gate?

DR. DILLMAN: He raced four times in one month.

QUESTION: Too many times?

DR. DILLMAN: I don't know.

DR. RAKER: This, I couldn't answer; I am not all that familiar with the case.

QUESTION: If this is an abscess, would you not expect more heat or swelling? In neuritis, we expect to detect a good deal more pain than this animal exhibited even when standing still.

DR. RAKER: The question is, in the case of an abscess, wouldn't there be more heat or swelling in the affected area or, in the case of neuritis, more pain or evidence of pain? Dr. Dey, do you care to respond to that question?

DR. DEY: My knowledge of nervous disorders is very limited but from the reading I was able to do on the way down, a peripheral nervous problem can involve either a lack of function, an increase in function, or a slight decrease in function. Therefore, if there is only partial involvement of the nerve supply, you may get an increase in the heat in the body; you may get a decrease, or it may remain the same. It all depends to what degree the autonomic nervous system is affected. The autonomic nervous fibers run with the peripheral nerve fibers and it depends upon the damage to these structures.

QUESTION: The horse was over-raced for thirty days. Is there not evidence that it was an acute affair, starting primarily from an acute deep injury, and apparently in the sacroiliac region?

DR. RAKER: Yes. I certainly feel this is an acute case and there is definitely injury in this area. Of course, I know the final diagnosis, but I think without it I would have felt the same way.

QUESTION: Any previous history of stifle trouble when racing as a two-year-old or three-year-old?

DR. RAKER: As far as I know, there has been no history of previous stifle trouble or lameness. Dr. Dey, would you care to comment on the stifle luxation? Any reasons for this?

DR. DEY: You can do a better job than I can.

DR. RAKER: Here is a horse showing marked, probably disuse atrophy of this leg, certainly there is a marked decrease in the size of the muscle along with a decrease in tone and function. In such cases, particularly where there is involvement of the quadriceps, I do believe one would often expect luxation of the patella as a secondary development.

Dr. Dillman mentioned that the horse sometimes developed a luxation of the right patella but that this was never a serious or severe affair. I would certainly anticipate this happening because I have seen it happen in other cases.

XUM

QUESTION: Can this increase in sensation to pin prick not be explained by the fact that this horse has an acute condition due to over-exertion with possible rupture of the blood vessels in the perineurium and calcification causing pressure on the nerve itself — something like a neuroma after neurectomy?

DR. RAKER: Yes. The question was, if I understand it correctly, whether this could not have been an acute traumatic lesion with hemorrhage, vascular damage and neural damage in the tissue. Is this correct, Doctor?

QUESTION: Right.

DR. RAKER: Yes, I think there are indications or signs that this very likely has occurred. As to how it occurred, there is no history of this colt having fallen, or being hurt in shipment, or anything of that sort. This is not to say it did not occur but we have no history of it.

Dr. Dillman pointed out last night that when he went to see the colt, everybody was ignorant; no one knew anything. Is this about right, Dr. Dillman?

DR. DILLMAN: Very, very vague.

DR. RAKER: They were very vague as to what happened, and so on. Maybe something did happen but we just do not have that information. I think this is a very good point.

FROM THE FLOOR: It seems that this horse was running quite a few long races over a fairly short period of time. Since tracks run counter-clockwise this might put quite a strain on the left hind leg and lead to neuritis in the femoral nerve, as Dr. Dey pointed out.

DR. RAKER: This is why we wanted to get the recent racing record of this colt and detect if the cause of the trouble might have occurred on the track. This is very true. You are saying that you feel it is a peripheral neuritis or nerve problem. Is this correct?

QUESTION: Involvement of the pelvic bones at all?

DR. RAKER: No evidence on standing or moving rectal examination by Dr. Jenny. I also asked Dr. Dillman and he found nothing abnormal in the circulatory supply or bony area of the pelvis.

QUESTION: The comment was made that you cannot have one part of the body affected with a central nervous disorder. I wonder if this is entirely true because following a stroke in man, one isolated limb can be affected very easily, due to stasis of the blood or stoppage of the blood in this area. I don't know if this is the case here but I did not want to let that point pass because you can get one limb involved with central nervous disorders.

DR. RAKER: Yes, I would agree and Dr. Dey would agree. I think what he was trying to point out was that it didn't appear to be of central or at least probably not of cerebral origin.

CHAIRMAN REED: I wonder if the trouble is not higher up than Dr. Dey suggested — possibly in the sciatic nerve, because of the atrophy that occurred higher than the extensor area itself?

DR. RAKER: You feel there is higher involvement basically?

CHAIRMAN REED: Not central nervous system involvement but there would be basically higher involvement in the area of the sciatic nerve.

DR. RAKER: And you base this on what?

CHAIRMAN REED: Primarily on the fact that the atrophy of the musculature is involved.

DR. RAKER: Dr. Reed says he feels (it really wasn't a question; it was a comment) that the pathology is probably higher, involving the sciatic nerve, because the muscles higher up near the midline are also involved.

CHAIRMAN REED: That is primarily right.

DR. RAKER: I will now give the pathologist's report. Dr. Craig destroyed this horse at the track and did a complete necropsy and he has given me a summary of the findings as noted on August 26.

"Grossly significant lesions were noted in the left sciatic nerve and its branches. The nerve trunks were surrounded by hemorrhage and inflammatory tissue, a reaction which appeared to follow the nerve trunks from the greater sciatic foramen to the hock, and which appeared to have spread along adjacent fascial planes. The superficial surface of the sacrosciatic ligament was markedly involved, as were the middle gluteal and pudendal nerves that traverse the ligament. Skeletal muscle masses served by the aforementioned nerve trunks were atrophic to a variable degree, with the most severe atrophy present in the middle gluteal muscle.

Microscopically, the perineural and fascial reaction consists of hemorrhage in various stages of organization. Marked fibrotic thickening of the perineurium and epineurium is present in the nerve trunks proper. Severe degenerative changes, predominantly of the Wallerian type, are distributed in patchy fashion throughout the groups of nerve bundles.

Further microscopic examination reveals marked neuronal degeneration and glial proliferation in spinal ganglia. The lumbar spinal cord is the site of a severe, subacute inflammatory response and associated degenerative changes. The myelitis is particularly severe in the ventral columns of gray matter and here is accompanied by neuronal degeneration and necrosis, and glial prolifera-

XUM

tion. Degeneration, primarily Wallerian, is also present in the radicles of spinal nerves that lie within the spinal canal." We have a few slides that will show some of these changes.

[Slide] The first slide is of a normal equine spinal cord. The photomicrograph demonstrates the spinal canal lined by ependyma on the left and a ventral gray horn in the right central portion of the picture.

[Slide] This photomicrograph demonstrates the intense inflammatory reaction, with attendant edema and softening, which was present in the ventral horn in the case in question. I think you can see considerable vacuolation and a general loss of density. In other words, this is edema and softening of the cord.

[Slide] The vertical collagenous bands in the left central portion of the photograph are the lamellae of the sacrosciatic ligament. Nerve radicles which course across its surface are seen just to the right, and are swollen and degenerated. Further to the right is seen a layer of granulation tissue and hemorrhage which was present on the superficial surface of the ligament.

[Slide] This section was taken from the sciatic nerve in the region of the femoral neck. Notice the intense fibrosis, granulation tissue and superficial hemorrhage which involve the nerve trunk and its sheath.

[Slide] This photomicrograph shows a nerve fasciculus cut in longtitudinal section, taken from a normal equine sciatic trunk. Notice the wavy, but evenly stained character of this group of nerve fibrils.

[Slide] By comparison, the sciatic nerve from the case in question shows marked swelling of the nerve fasciculus, with irregularity and vacuolation of the myelin sheath that surrounds individual fibers. Axons are variably swollen, fractured or altogether absent.

[Slide] This shows a normal sciatic nerve fasciculus, cut in transverse section.

[Slide] In this slide, compare the affected nerve cut in transverse section. The perineurium is distended and the space around the nerve bundle contains proteinacious material. The fibrils within the fasciculus are markedly degenerated, as evidenced at this power by the distention and swelling of the myelin sheaths.

DIAGNOSIS: **Severe subacute myelitis, lumbar cord, with associated peripheral neuropathy.**

I think you gentlemen all did very well.

QUESTION: I was wondering if any pathogenesis could be explained in this case.

DR. RAKER: I believe it is definitely of traumatic origin and probably associated with the stress of racing. Whether this horse

actually slipped or not, we do not know. A better history might be helpful but we don't have it; so much for the first case.

The second case is with reference to a two year old standardbred filly. I have seen this case; in fact, I admitted it into the clinic, and eventually it ended up in the hands of Dr. Jenny who did some surgery on it.

On August 24, 1961, a two year old standardbred filly was referred to the Equine Clinic, University of Pennsylvania School of Veterinary Medicine, with a chief complaint of a hard, progressive swelling on the inside of the left forearm about four inches above the carpus.

The history stated that the swelling had first been noticed in the spring of 1960. It had progressed over a period of several weeks to a size of approximately 1 x 2 x ½ inches. The enlargement became static at that size and lameness was never noted. In the spring of 1961, the filly was put into training and trained well until toward the end of July. At that time, she became slightly lame in the left foreleg and an increase in the heat, tenderness and size of the swelling was noted, beginning at about that time and progressing to the present size of 2 x 3½ x 1 inches.

A physical examination revealed no generalized or remote symptoms which might be referred to the chief complaint. Examination and palpation of the involved area revealed a bluntly fusiform, apparently bony enlargement of the medial surface of the radius. There was a slight increase in local temperature and mild sensitivity to vigorous pressure over the area.

Radiographs of the area revealed an intense sclerotic fusiform thickening of the radial cortex with a slight indentation at its most prominent subcutaneous point, and faint locular lucencies were noted in the interior of the thickening (Fig. 1).

On the basis of our clinical diagnosis and the history, the decision was made to attempt surgical removal of the lesion.

Now I am going to turn this over to Dr. Milne.

DR. MILNE: During our conferences in Canada which we have every week, I have the unfortunate habit of trying to jump the gun. I get the history, I say "Yes, that is what it is". Then I get red-faced when the diagnosis is given!

I jotted down a few possibilities, and the first one is periostitis from injury. Here is a standardbred. We might think in terms of conformation or rather, poor conformation and the possibility of cross-firing; it is a little bit high up the leg however, for that. Apparently, there is no history of any previous injury.

Then, of course, we go to the more bizarre complaints. Could this be an abscess — Brodie's abscess? To my mind it does not seem to fit the pattern that one would expect from a classical Brodie's abscess.

XUM

Then, could it be neoplastic? Here is a young horse, two years of age (and I know we get tumors in young animals) but as far as I can see, this does not just seem to fit into the pattern of fibro-sarcoma or osteo-sarcoma, but I am not smart enough to tell how one could differentiate osteochondroma on an existing bone. Could it be low-grade bacterial infection or involvement? There is apparently the history of a hard fibrous mass but no fluctuation, indicating abscessation of soft tissues. Some of the bumps felt on animals are extremely hard, yet on x-ray we find bone is not involved, only soft tissue. The x-ray clearly points out that there is bony or at least, periosteal involvement. I cannot help but insert something of a humorous vein here. I wonder if Dr. Mason's prickly pear could have had something to do with this? Maybe they don't have that type of fruit in the region where this horse comes from!

My own feeling on this, and here is where I am sticking my big neck out — is this a foreign body reaction? The favorite story we get is that someone slugged the horse with a .22 rifle. There can be a foreign body such as a tiny sliver of wood which may be extremely hard to pick up on x-ray. This could be such a case so just to cut the thing short and throw it open to discussion, I will go along with foreign body reaction, chronic since the condition was noticed in the spring of 1960, and it has been there for sometime. So, can we throw it open at this point?

DR. RAKER: Well, I think that was very good because Dr. Milne certainly has not had much chance to go over this. His diagnosis basically is — correct me if I am wrong — an osteitis probably due to foreign body? He shakes his head "yes" that this is his clinical diagnosis.

Now do we have any questions from the floor? Anyone have any ideas? Anyone have any questions?

DR. C. H. REID: Do they use steel brushes?

DR. RAKER: I would say no but I am not sure. From the management program as I saw it, when we talked to the owner, I would rather doubt it.

QUESTION: Not to confuse the issue — I cannot see the radiographs too well — how about osteogenic cyst?

DR. RAKER: The doctor asked about the possibility of osteogenic cyst. Dr. Milne, care to comment on that?

DR. MILNE: I think the fairest thing would be to pass the x-ray down to him. It could possibly be. Charlie knows but he is not telling.

DR. O'DEA: Was this filly a trotting or pacing horse?

DR. RAKER: Pacing.

DR. O'DEA: What was the situation on hobbles?

DR. RAKER: There were no hobble burns.

DR. O'DEA: You did not see her in the initial stage?

DR. RAKER: I did not see her when this occurred.

DR. O'DEA: In the standardbred we see quite a number of subperiosteal hematomas that calcify. The other thing, if it was traumatic, there is a possibility of there being a very, very small bone spicule, a bone sequestrum which might be the foreign body situation that Frank is thinking of — subperiosteal; sequestrum of a little chip off the cortex.

DR. RAKER: We have seen some of these hematomas which organize, and I concur with the suggestion. The feeling is here, however, that there is definite evidence of some tract through the bone, some demineralization which maybe you could not see too clearly on the screen but it does show on the original film. I think that is a very good suggestion.

The clinical diagnosis was **SCLEROSING OSTEITIS** and Dr. Jenny operated on the case. The surgical and post-surgical findings are as follows: "A scar was noted over the eminence when the hair was clipped". This we did not see clinically; we did not know of it until we clipped the hair off. After reflection of the skin, a small sinus in the center of the lesion was accidentally opened and yielded a drop of greenish-yellow pus. During the course of chiseling out the sclerotic bone, several similar pockets were encountered in the depth of the cortex.

After nine days in culture media, a sample of the pus yielded Corynebacterium pyogenes, highly sensitive to penicillin.

A histological examination of the bone removed at surgery, revealed a fusilorm lamina of dense, mature bone overlain by active periosteum and including foci of immature fibro-vascular connective tissue infiltrated with large numbers of polymorphonuclear leukocytes.

FINAL DIAGNOSIS: **INFECTIOUS SCLEROSING OSTEITIS.** The wound healed uneventfully and a six-weeks' followup has not yet revealed any evidence of recurrence. This is, we feel, too soon to say that it will not recur.

Do you have any other comments?

QUESTION: I would like to ask Dr. Carlson what type of results he would expect from radiotherapy on this case.

DR. RAKER: The Doctor would like to direct a question to Dr. Carlson as to what results he might expect by exposing this to x-ray therapy. Would you care to comment on that, Dr. Carlson?

DR. CARLSON: We haven't had a chance to do much work on this type of thing. I think surgery is indicated, first and foremost. I think that probably would clear it up. X-ray therapy would theoretically work but surgery first.

XUM

Clinico-pathologic Conference; Fig. 1. Faint locular lucencies are noted in the interior of the thickening.

FOLLICLE EXAMINATION OF OVARIES IN RELATION TO OPTIMUM BREEDING TIME

J. D. Gadd

The purpose of this paper is to offer statistical evidence of the efficacy of follicle examination of ovaries for optimum breeding time.

This year I was afforded the opportunity to follicle-examine every mare except one, that was bred to Native Dancer. I had been using this technic for 3 previous seasons, but only in limited form, and hence without full records. It is appropriate at this time that I give credit to the original work of Day, of Newmarket, England, (Veterinary Record, Vol. 69 (1957) L, p 1258).

When I first learned of Day's work, I asked Dr. William R. McGee of Lexington for his evaluation of its probable usefulness in the United States. I well remember that he said, "You will be able to obtain better results than I, having all your mares on one farm, for you will be able to follow them more closely." This I have found to be true.

At the same time, Dr. McGee presented me with cards on which I could record daily the degree of response to teasing, the degree of estrus in numbers and the size and consistency of the follicle. Printed on the cards were outlines of the 2 ovaries. On these outlines I recorded the size, number and position of the follicles each day. These cards, carried in a small loose-leaf ring binder, were filled in after the examination of each mare. There was a certain amount of human error, for I sometimes forgot the book or failed to record my observation until 2 or 3 mares had been examined. I therefore found it imperative to record my findings immediately after each examination.

The mare does not have the defined, rhythmic, reproductive cycle seen in laboratory animals, in the cow and in the human. Estrual behavior varies according to environmental factors. Sexual development commences with the beginning of spring and the increase in hours of daylight. Early testing for establishing a breeding cycle is one aid. The estrogenic content of new budding grass is another. Many other factors tending to increase metabolism are contributors to sexual development.

All the reproductive organs are in constant change as one estrus follows another and cycle follows cycle. Under the influence of the ovaries, which in turn are controlled by the pituitary gland at the base of the brain, a recognizable series of events takes place in the entire genital tract at intervals of about 3 weeks. These

XUM

events coincide with recognizable changes in the size, shape and content of the ovaries.

Examination of the mare's ovaries reveals the size of the follicle. At the beginning of the estrual period you may feel 2 or 3 small follicles. Typically, one of these develops quickly to a larger size. It first shows a tenseness, next a softness, then a stage in which fluctuation is felt between the palpating fingers. This fluctuating stage represents the optimum time for breeding: the follicle is ready to rupture and ovulation occurs.

The follicles vary from 2 to 4 centimeters in diameter. A small ovary will have a small follicle; a larger ovary will produce a comparatively larger follicle.

The follicle may develop in any part of the ovary. Two may develop together, or one after another a day or days apart—which often may account for longer heat periods. When you palpate 2 large follicles, whether in the same ovary or one in each ovary, you hope that one of these will regress and that only one mature ovum will be released, since twin ovulation predisposes to twin conception. Day reports that he sometimes allows one follicle to ovulate, then breeds on the second follicle, giving a luteinizing hormone to ensure ovulation. My conservatism limits me to advising a single breeding in that heat.

There is one type of follicle development that I have not seen recorded. It is what I call a "meaty" type. It forms in the middle and base of the ovary and develops in a cyst-like formation. When it ruptures, almost the entire ovary is evacuated, leaving a comparatively huge crater. I was able to recognize this "meaty" or "doughy" kind of follicle before ovulation. I found 3 of them this year in 3 different mares and in no instance did the mare conceive in that heat period. Two of these mares conceived in the next period, each of them ovulating from the same ovary which had produced the cyst-like follicle. The third mare in this group had a spurious conception.

I have used more luteinizing hormone this year than in all my previous years of practice combined. I do not believe in using it as a routine, but used it in selected cases, such as barren mares observed early in the season with small, irregular follicles like grape clusters. These mares are not covered until the grass begins to come, when they show better under speculum examination. If a follicle is heading, a larger than normal dose of chorionic gonadotropin (3,000 units intravenously) is given on the day of breeding or the day after. I follow the same pattern whenever luteinizing hormone is used, to minimize the chance that the follicle will rupture before the mare is bred. Normally I use 1,500 units of chorionic gonadotropin as a minimum dose. I believe it has been helpful and in cases where it failed to result in conception, it may

have stimulated estrogenic activity in the ovary, since the mares often conceived in the following heat period.

I examined each mare on the second day she responded to the teaser, and at the same time made a speculum examination of the vagina and cervix to ascertain the degree of coloration and relaxation and whether the membranes were dry and gummy or glistening and fluid-like. Then the mare was examined every other day, or even daily if the follicle was maturing rapidly or there was a question as to the situation.

It was not unusual to find, on the first examination, a mare in evident estrus and with a large follicle that fluctuated under palpation, and if the results of speculum examination and teasing were confluent, the mare was bred immediately.

There are many variations from the typical picture. Early in the year the heat periods usually are longer and the follicles slower in developing, and estrogenic activity has been insufficient to develop the uterus, cervix, or vagina to such a point that the mare would be receptive or would conceive, even if covered. There is also what I think of as a psychological or nervous heat, in which the mare shows to the teaser but is negative to speculum examination, and no follicle is found in the ovary. In this type of long heat, when the mare suddenly softens and relaxes in the presence of the teaser, the examiner may find a ripe follicle and conception may occur if the mare is bred that day or within 48 hours of ovulation.

Breeding immediately after speculum examination is not recommended, since the inflated vagina decreases the stallion's libido. The condition may be minimized by manual pressure per rectum to deflate the vagina and by turning the mare out for 15 or 20 minutes before she is brought to the breeding shed.

Some idea of the mare's readiness for service may be derived from speculum examination; a fluid-like appearance of the vagina and relaxation of the cervix usually are coincident with maturity of the follicle. There are, however, variations and inconsistencies in the pattern and it is for this reason that rectal palpation of the developing follicle provides a more accurate estimate of the optimum time for breeding.

It is my observation that the egg normally is shed 24 to 48 hours before the end of estrus. Thus it is advantageous to examine the mare 48 hours after breeding. If the follicle is still present there is the opportunity for a double cover.

On theoretical considerations, service just before or after the time of ovulation is regarded by many as giving the optimum chance of conception, but, according to Belonje, evidence is accumulating that the fertility percentage is considerably increased by insemination up to 12 hours after ovulation. This year, I had

XUM

4 mares that conceived as the result of covers made after what I had recorded as partial rupture of the follicle. Breeding at this stage had not been contemplated in the original design. The mares were examined and covered the same morning because we did not want to miss the chance of conception in that heat. I merely recorded my observations, saying that I doubted they would conceive.

Afterwards, on the basis of Belonje's work, I realized that I must have recorded recent ruptures as partial ruptures. The observations which must have been made less than 12 hours after ovulation, revealed a break or rent in the capsule; there was no palpable fluctuation, and it was still too early to feel the crater yield to pressure in the typical "snowball crunching" manner or to find evidence of the blood clot which later is invaded by blood vessels and becomes luteinized. The size of the subsequently-formed corpus luteum was usually less than that of the Graafian follicle, but sometimes approximated it. The corpus luteum remained palpable for 2 or 3 days*, and when the ovary was handled there was evidence of low-grade pain, indicated by lifting of a leg. It is useless to breed after complete rupture of the follicle.

As the genital tract of the mare is still under the influence of its own estrogens in the period of estrus, the defense mechanism is highly organized. The sperms in the uterus are stimulated to greater activity, then die and begin agglutinating and are finally removed by leucocytic action. Slight reddening and swelling, with desquamation of the superficial epithelial layers (resembling a mild inflammation) take place within 8 hours of insemination. Antigens are produced which, if in high concentration, kill off the spermatozoa. This is the normal defensive reaction against infection which might be caused by organisms introduced at copulation. It constitutes a reason to avoid repeated covers at short intervals during the heat period.

The principal types of abnormal cycles observed at stud farms year after year are:

1. Anestrus, or lack of heat. The barren mare goes through most of the breeding season without evidence of estrus. The foaling mare may be "out" for an extended time after the "foaling heat" period.

2. Anovular heat. The mare shows normal symptoms of heat, but fails to ovulate. This condition I have found to be more prevalent than I had ever imagined before I began making follicle examinations. The follicle sometimes became quite large in comparison with the ovary, but a day or so later it had regressed.

*EDITOR'S NOTE: Most authorities are of the opinion that in the mare the corpus luteum is not palpable at any time of the cycle. It is presumed that the author is referring to the corpus rubrum or blood clot which is the precursor of the corpus luteum.

This observation was made in the course of re-examinations to determine whether a double cover was indicated.

3. Estrus without follicle development. Mares show external signs of heat and receptivity, but rectal palpation discloses no follicle formation.

4. Irregular estrus. Cycles vary within the season and from year to year.

In "silent heat" there is good follicle formation; the mare does not respond to the teaser, but may show to the speculum. Some mares may show to a different teaser or to the stallion, while remaining cold to a familiar teaser. When speculum examination is in accord with follicle formation, many of these mares may be hobbled and bred without difficulty. Usually they are quite willing to accept the stud.

A prolonged heat is one in which a mare stays in heat for 20 to 30 days and is receptive to the stud at any time during this period. Such mares may become pregnant when adequate follicle formation is observed. They should be bred within 48 hours of the predicted time of ovulation as determined by ovarian palpation.

As implied by the list of abnormal types of cycles, there are numerous different combinations of behavior. There may be estrus without follicle formation; estrus with follicle formation, but without ovulation; or follicle formation and ovulation without estrus.

MISCELLANEOUS OBSERVATIONS:

Of 17 mares bred on the ninth day or foaling heat, 15 had been examined for follicle formation. Six conceived, including one bred on the 11th day after foaling. One mare was passed over at first because she had a ruptured follicle and also because she had had a heavy placenta, but on the 17th day after foaling, when she was normal and receptive and had a ripe follicle, she was covered and conceived. The other 8 mares failed to conceive in the foaling heat.

Three mares had spurious conceptions. Two of them conceived after being induced into heat and examined for follicle formation.

Four mares conceived as the result of covers made after the follicle had ruptured. Presumably the covers were made less than 12 hours after ovulation.

Four mares conceived when the follicle ruptured more than 48 hours after cover. One follicle was still present 5 days after cover; for that I have no explanation.

Four mares went through heat periods without being bred,

XUM

because the follicle had ruptured. One of these was on the ninth day after foaling. Another mare was in heat only 2 days.

Two mares conceived from single covers on the second and third days of short heat periods in February. Ordinarily the heat periods are quite long early in the year, and it is best to ignore them unless the optimum time for breeding is established by follicle examination.

Two other mares conceived despite the fact that urine was found on the floor of the vagina towards the end of 4- or 5-day heat periods. Without the aid of follicle examination, both would have been passed over.

One mare had been barren for 2 years and her condition had not improved, so I resorted to radical means; for the first time, I applied a deep perineal suture in the breeding season. The deep suture was done on April 1. The mare was in season 14 days later and was in such good condition that she was covered and became pregnant.

Only 2 mares were bred 3 times in one period. The follicle persisted, and the stallion was not busy. Both conceived. One of these aborted in October; my records showed that a follicle had been present in each ovary on that one mare.

One mare went through 8 heat periods, but conceived.

Of the 4 mares which proved barren, 3 were known to be poor breeding risks. Excluding the 3 problem cases, I must conclude that 35 mares were settled out of a total of 36 which might have become pregnant.

I have made the assumption, without experimental or statistical evidence, that stallions get better offspring when they are not used excessively, since the sperm should be stronger physiologically and perhaps genetically. The stallion which begets an outstanding horse as a result of covering a few mares early in life may sire nothing of high class later in life when he has a full book. I have observed numerous instances of such a pattern. Whether this observation is valid or not, there are many other reasons for follicle examination under circumstances where this technic may be employed on a routine basis.

Apart from examining mares for abnormality or disease, when we find 4 or 5 mares in season at once, it is helpful to be able to determine by follicle examination the most likely prospect for succeeding covers.

Ovarian palpation frequently provides an explanation for infertility. It sometimes enables corrective measures to be taken. It enables mares to be taken at the optimum time for breeding. It allows the stallion to be saved when conception is impossible. Finally, it results in more early-season conceptions and thus allows more latitude for problem cases and for the peak periods of

demands upon the stallion. It should be especially useful in dealing with the less virile stallion.

DR. CARRICABURU: Could I have the reference to Belonje's work?

DR. GADD: It is The Physiology of Reproduction in the Mare with Special Reference to the Thoroughbred, by C. W. A. Belonje, Journal of the South African Veterinary Medical Association, Pre-Congress Number, March 1960, page 115.

QUESTION: I wonder if you would comment on the approximate size of the follicle about to rupture, also the location; in other words, was it distinct from the ovary, or incorporated in the ovary? Third, your experience with manual rupture of the follicle at that time.

DR. GADD: One at a time.

QUESTION: First, the approximate size of the mature follicle?

DR. GADD: A follicle that is going to rupture, varies in size. Some of them are bigger than the ovary itself — these are amazing, but do not form the typical picture. They are 2 to 3 centimeters in diameter. If you are able to follow them over a couple of days, you will feel them develop, but you cannot always do that. Sometimes you have to size them up that day. If they seem good and ripe, in other words, fluctuating, breed her.

QUESTION: Is this follicle incorporated in the ovary or distinct from the ovary?

DR. GADD: No, it is incorporated in the ovary. It starts in the body of the ovary, enlarges, forms a small cyst-like formation, and gets bigger and grows out over the surface.

QUESTION: You can then palpate two distinct lobes?

DR. GADD: You might palpate only one lobe; there is only one mature follicle.

QUESTION: In the cow you can almost feel a separate follicle-ovary pattern where there is a follicle and the ovary is on the end of it.

DR. GADD: The follicle is part of the ovary. The ovary does not enlarge; it is the follicle. It is a cyst-like formation. It is like the one developing in a cow, except that it is bigger.

QUESTION: What is your experience with manual rupture of the follicles?

DR. GADD: Only rarely can a follicle in a mare be ruptured and I don't try to rupture them. I have felt that I had possibly ruptured one or two but I think they would have ruptured in a matter of an hour or two, anyway.

DR. MOORE: Your deep perineal suture, could you give a quick technic?

XUM

DR. GADD: On the dorsal roof of the vulva, I dissect the mucous membrane down at least a third on each side so that the whole thing is denuded back for 6 inches. The idea is to take off the mucous membrane and then with interrupted sutures, pull the dorsal wall of the vulva in. This helps to stop the aspiration of air into the vagina. I do this only on mares where the labial suture does not work.

QUESTION: Could you give us a little more information on the weight of the placenta relative to breeding her on the ninth day?

DR. GADD: This was an observation of Dr. Caslick back in 1930. It was routine to weigh all the placentas, and it is also the practice of all of us to have the placenta saved for us until the next morning and spread out, so we can ensure that the entire afterbirth is present. We can tell more by that than we can by manually examining the uterus.

QUESTION: Will you say something about the afterbirth if it weighs over 14 pounds?

DR. GADD: If it weighs over that, it shows there is infection, and it is better to let them go over the ninth day. That is an observation of Dr. Caslick and most of us follow it. For instance, we had one mare with heavy placenta, and she did not clean.

QUESTION: Do I understand it is a regular practice to not breed if the follicle has ruptured? Is that right?

DR. GADD: That is correct, but I cite these five instances where I found the follicle had just ruptured, or five mares where the follicle had just ruptured, and they conceived. So, if you just feel a break and it is not a typical fluctuation but you can still feel the membrane, then you know you are within 8 to 12 hours, and it is all right to breed them. Ordinarily, if the follicle has ruptured, they are not bred. That is the observation of Day, and I agree with him.

DR. JOHNSON: I wonder if you would comment on mares with flooding of the vagina with urine. What is your approach in such cases?

DR. GADD: Up until this year, I felt it was useless to breed those mares, because there would be inflammation of the vagina; many times the cervix will be relaxed, and there is very little chance of conception. Hagyard has swabbed them off and gone ahead and bred them. What his results are I do not know. I do feel that conception is possible if, sometime later in the heat period the mare is receptive and I did not find the urine present; some of these conceived.

Another extreme case that I did observe (not in this series), was a mare examined late in the heat period. She had been bred twice. When I examined her, there was urine on the floor. I said,

"There is no use breeding this mare. I just wouldn't fool with her". She was a cheap mare. They didn't breed her. I examined her in October and she was in foal. So, the more I do, the less I know!

DR. CARRICABURU: About the double and triple covers, at what intervals would you make the covers — 48 hours?

DR. GADD: The routine procedure is to breed them at 48 hours, because we feel that they may conceive within 48 hours of the follicle rupturing. Therefore, we breed at 48 hours. Then, if they are still in season, I let them go three days after they have been bred the second time: two such cases conceived on the triple cover.

QUESTION: You say if the follicle has ruptured, you don't breed, yet, on the other hand, you say that you feel you will get conception within 48 hours on double cover. Isn't it likely that, if you examine every two days, and in the interval the follicle has ruptured, you can go ahead and breed, and probably get conception? Or do I misunderstand that?

DR. GADD: I am not quite sure, Doctor, but evidence has been accumulated to show that, if you breed the mare 12 hours after rupture of the follicle, she will conceive. It is a pretty fine technic to be able to tell in these mares if the follicle has just ruptured, and I have tried to describe what I feel is the appearance of a "just-ruptured" follicle. Also, in my statistics I uniformly called them "partial rupture". I did not know what I was finding until I read Belonje's report and made the observation that those mares conceived.

QUESTION: What type of luteinizing hormone do you use?

DR. GADD: I use Upjohn's gonadotrophin. CHORONIC

QUESTION: I would like to know again when you use ~~chronic~~ gonadotrophin.

DR. GADD: I use it on any problem mares. It is a luteinizing hormone. I never use it until after the mare is in season. In other words, you use it to get the follicle to rupture, not for development of the follicle. That is an important difference. Use it on mares after they have come in season, the follicle is present and you want to get it to ovulate. Some people have used it on every mare and in addition, have given too much of it. So, don't go overboard on it; don't try to use it on every mare. Bill McGee tells me that laymen have used it on some farms and they have run into trouble.

QUESTION: How do you stimulate follicle development? May I clarify that? A mare comes in season and has an early follicle development and then becomes static, how do you proceed with those?

DR. GADD: Actually, I don't use anything; I depend on teasing. Our method of teasing is so good that, if the mare does not come in season that heat period, when she should, we might go to the speculum. I do not use any hormones here.

XUM

QUESTION: After the mare is in heat, you still don't use anything to mature the follicle, to change it to a ripe follicle?

DR. GADD: Yes, that is what I have been talking about. I use the luteinizing or chorionic gonadotrophin. That is not for the development of the follicle.

QUESTION: I mean for development.

DR. GADD: After the follicle is present, we use that, just to get it to rupture.

QUESTION: Ever used Armour's luteinizing hormone?

DR. GADD: I have never used Armour's luteinizing hormone. You use one type and I use another; you take your pick.

FROM THE FLOOR: The reason I raised the question is that if it is desired to end heat in a filly, I find one vial will not do it — yet three will.

DR. GADD: He said that his treatment for these fillies that stay in heat too long, was to throw them out of heat by using a triple dose of luteinizing hormone. That is all right.

QUESTION: What do you do with mares that stay in prolonged heat after breeding?

DR. GADD: We keep right on teasing them. We don't try to change them. But, for instance, if the stud man says, "This mare is hotter today, would you look at her?" and you find the speculum examination is consistent with what the teaser reveals and you also find a good follicle is getting ready to rupture, breed.

QUESTION: Then she stays in heat?

DR. GADD: You might breed her a second time, but I haven't done it.

QUESTION: Just ignore her?

DR. GADD: Yes.

FROM THE FLOOR: I have had some mares stay in as long as three weeks.

DR. GADD: Often, it is common. We all experience that.

QUESTION: What about a mare that runs about a 12- or 14-day cycle and the ovary varies from golf-ball size to goose-egg size, and the mare goes completely out of heat? She never shows a good heat but will always show somewhat to the stud.

DR. GADD: This is what we speak of as a long heat period. Everything is important there. If she relaxes and shows better to the stud, and the rectal examination reveals that the follicle is good and ripe, breed her.

QUESTION: That is not quite what I was getting at. The ovary runs about a 12 or 14-day cycle, varying from those two different sizes. The mare stays in all the time, over a period of months — never goes out.

DR. GADD: I do not depend on the ovary, I would try and determine what the follicle is doing.

PERIODIC OPHTHALMIA

W. G. MAGRANE

The purpose of this paper is to review briefly the work of many men as regards possible causes; to consider some recent research into the cause and histopathology, and to discuss medical and surgical treatment that has been proved of value.

SYNONYMS

Moonblindness, recurrent uveitis, anterior uveitis, recurrent ophthalmia, recurrent iridocyclitis, periodic iridocyclitis. The term periodic ophthalmia is certainly not descriptive of the pathological processes embracing this disease. It would seem that either recurrent iridocyclitis or uveitis would be more appropriate names for this condition since it would indicate more specifically the actual pathology involved—a recurring inflammation of the iris, ciliary body, and eventually, the choroid (uveal tract).

CLINICAL FEATURES

Photophobia and lacrimation are seen early, often with an accompanying eyelid edema and catarrhal conjunctivitis. Other early findings include ciliary injection, miosis, deep keratitis and congestion in the retina.

A cellular exudate often occurs in the anterior chamber of the eye on the second day of an attack. This is usually flocculent and gravitates to the lower portion of the chamber, but there may be merely a diffuse cloudiness. Systemic changes, when present, include dejection, inappetence and fever.

Later symptoms are more severe. Photophobia becomes very pronounced as an attack progresses. Cloudiness of the transparent media occurs and the vitreous humor may contain patches of opaque material. There is commonly a reduction in intraocular pressure causing a characteristic wrinkling of the skin of the upper eyelid which persists even throughout the quiescent stage. Usually these acute symptoms disappear during the quiescent stage; the exudates are absorbed, and transparency is restored to the media. After several attacks, however, complete recovery does not occur; residual lesions become more and more severe until blindness ensues. Posterior synechia frequently results in tearing, atrophy and distorsion of the iris. Retinal detachment, dislocation of the lens, cataract, and phthisis bulbi are often the final result.

We have, then, the classical picture of a pure iridocyclitis which in turn may lead to numerous complications and sequelae. Regardless of species, iridocyclitis presents similar findings and I have had occasion to observe the condition in man, the dog and

XUM

the cat. A comparable type of recurrent exudative uveitis continues to plague many human individuals. Clinical and laboratory research into this disease is continuous and has become a full time project for many ophthalmologists.

Editor's Note: At this stage of the presentation, Dr. Magrane showed excellent color slides of the following conditions: — iritis, deep keratitis, hypopyon, exudate on anterior capsule of lens, synechia, iris atrophy, cataract and sub-luxated lens, retinal detachment and phthisis bulbi.

FUNDIC FINDINGS

During attacks of periodic ophthalmia, photophobia and opacities in the ocular media interfere with examination of the fundus. Following attacks, corneal scars, pupillary adhesions and membranes, or lens and vitreous opacities also often preclude a satisfactory examination.

Recently, Dr. S. R. Roberts of Richmond, California, spent a year in ophthalmic research at the Veterinary College in Vienna and will shortly contribute to the world literature, more information regarding the fundic lesions associated with this disease. In brief summary of his work "a circumpapillary choroiditis was found in the eyes of 35 horses out of 456 examined. The lesions were pale areas on one or both sides of the papilla in which pigment streaks were found. Of 15 eyes examined histologically, 10 exhibited inflammation of the uvea (focal chorio-retinitis). In the other five there was no sign of active inflammation so these may be late or "burned out" stages of circumpapillary choroiditis."

ETIOLOGY

The etiology of this condition has been the subject of much speculation and relatively unrewarding research. The etiology is unquestionably a complex one, involving many factors, and, certainly, has not yet been satisfactorily settled. Throughout the studies on etiology, workers would seemingly find the answer — only to have some new aspect turn up to further cloud the picture. The very nature of the disease, the fact that it is but a manifestation of another disease rather than a specific entity in itself has made etiological studies difficult.

In the recorded literature, we find studies of the etiologic relationship of geographic, climatic, genetic, nutritional, toxic, bacterial, viral, parasitic, hypertensive and other influences to periodic ophthalmia.

HEREDITY

Early 20th century workers speculated on the possibility of a hereditary predisposition or susceptibility. More critical examination of breeding records and statistics shows heredity to play only a minor role, if any, in this disease.

HYPERSENSITIVENESS

Hypersensitivity to various agents has been considered as an etiological factor. Toxins elaborated by internal parasites, products of protein decomposition, toxic protein products, and foreign proteins such as tuberculin may all affect the eye. The possibility of sensitivity playing some part in this condition should not be overlooked. More frequently, sensitivity phenomena are being demonstrated to have a relationship to the pathogenicity of various infectious diseases.

NUTRITION

Jones and others, reported success with riboflavin in the prevention of periodic ophthalmia. Still others have reported trouble with periodic ophthalmia on farms where riboflavin has been added to the ration for several years. It is generally thought that riboflavin has no more prophylactic value in this disease than an adequate diet has in the prevention of almost any disease, and that its use will not alter the course of established cases.

VIRUS

At one time a filterable virus was suggested as the etiological agent. In 1930, Woods and Chesney succeeded in transmitting the disease, using filtrates of ocular fluids and tissue from affected eyes. Stubbs et al, in attempting to confirm this work, had moderate success, but the significance of this success is questioned by themselves and others because of the development of symptoms in several control animals not receiving the filtrate.

PARASITES

A number of parasitic larvae have been found in the eyes of horses with ocular conditions of one sort or another but the symptomatology in these cases was not that of typical periodic ophthalmia.

Roberts, however, in his recent study in Vienna, observed in 4 eyes, parasitic bodies identified as microfilaria of Onchocerca Cervicalis in the superficial corneal and conjunctival layers, together with a marked lymphocytic reaction. He suggests that these parasites may be etiologically related to periodic ophthalmia.

BACTERIA

Numerous workers have tried to incriminate many organisms but further work in each case revealed that the organisms studied were either secondary invaders of a non-specific nature, or normal, non-pathogenic bacteria commonly found in the eye. Brucellosis, leptospirosis and streptococcic infections have all been suspected.

Much work was done with Brucella organisms after it was suggested that this organism might be the etiological factor. Other

XUM

workers then demonstrated that no correlation could be established between brucellosis and periodic ophthalmia, as measured by agglutination tests.

Heusser began his work with leptospirosis in 1948 and after exhaustive studies of his own and reviewing the work of others, concluded in 1952 that periodic ophthalmia is not a specific disease entity in itself, but rather, a residual iridocyclitis remaining after a generalized systemic leptospirosis; the ocular disturbances occurring at a variable time after the generalized infection. He further stated that periodic ophthalmia need not be a constant sequel to all cases of equine leptospirosis, and expressed the opinion that no cases occur without a previous infection with Leptospira. Also, his experiments did not demonstrate any relation of periodic ophthalmia to riboflavin deficiency.

In 1954, Witmer, a physician, supported the work of Heusser, and said there was little doubt in his mind as to the etiology of this disease. He pointed out that more and more human leptospiral uveitis is being reported. An interesting fact uncovered in his study of the horse was that during the acute stage, especially in later relapses of the disease, the agglutination titer in the aqueous humor is often much higher than in the serum. He suggested two possibilities for these relatively high agglutination titers in the aqueous humor: (1) it is conceivable that the antibodies are transported from the blood by an increased permeability due to the inflammation; (2) it is also possible that the antibodies are formed locally in the infected tissue. He then went on to conclude that their findings support the suggestion that periodic ophthalmia in horses is largely a local leptospiral infection. The local and selective antibody formation in the inflamed eye can cause very high agglutination titers.

In an addendum, Witmer then revealed several striking facts:—

All horses imported from Germany into Switzerland were suffering from Leptospira Grippotyphosa. The only affected Swiss horse was suffering from Leptospira Pomona. Practically all horses had streptococcal infection during the acclimatization period. He stated, however, that this seems to be a general rule for all imported horses. Fresh cases show low titers; old cases, after several relapses, show very high agglutination titers.

It has been suggested by others that periodic ophthalmia is due to a reaction of the uveal tract to antigens from repeated infections, usually of a streptococcal nature.

Personally, I do not intend going out on a limb to offer any conclusions. The fact remains, however, that in periodic ophthalmia we are dealing with an absolute iridocyclitis, and that iridocycli-

tis in man, the dog and the cat is ordinarily due to some systemic foci of infection. There is, therefore, no reason to doubt that its occurrence in the horse is secondary to a comparable infection — whether it be leptospirosis, brucellosis, streptoccoccal or other infection.

TREATMENT

There is a pronounced absence of any controlled experimental work on the treatment of this disease. This is understandable in the light of the fact that the etiological factors are obscure.

Local treatment is symptomatic in nature. Atropine (2%) should be used in every instance to keep the pupil dilated and aid in the prevention of posterior synechia. Its usefulness goes beyond that though, as atropine paralyzes the ciliary body, thus alleviating ciliary spasm, an important source of pain. Atropine should be used hourly until dilatation is achieved and then twice daily.

Corticoids are very helpful but must be used by systemic and/or sub-conjunctival injection to be effective. Topical application, combined with an antibiotic, may also be used, but in this form its activity is confined to the anterior segment of the eye.

Under adequate tranquilization and topical anesthesia, up to 20 mg (1cc) of Hydeltrone T.B.A. (Merck), or a similar prednisolone substitute, may be injected under the bulbar or palpebral conjunctiva. In the equine, the palpebral is the most accessible. On withdrawal of the needle, a twisting action will prevent leakage. This injection should be repeated at 48 hour intervals. Systemic dosage of a corticoid, would of course, depend on the weight of the horse.

Systemic use of an antibiotic would depend on ascertaining the focus of infection. If a fever is present and leptospirosis thought to be the cause, dihydrostreptomycin has proved helpful.

It must be remembered, however, that if a local effect on the uveal tract of the eye is desired from the use of systemic antibiotics, only chloromycetin and the sulfonamides are capable of penetrating the blood-aqueous barrier and reaching the area.

Paracentesis of the anterior chamber is a most useful procedure if hypopyon is present. Besides releasing the exudate, the secondary or plasmoid aqueous which replaces that drained from the chamber, is high in antibody titer and most beneficial to the disease process.

In spite of proper, early and satisfactory treatment, we are plagued with the knowledge that future attacks are impending. This led Dr. J. M. Dimic of Yugoslavia to a fascinating piece of research on the surgical treatment of periodic ophthalmia. In the

XUM

Proceedings of the XVth International Veterinary Congress, Stockholm, 1953, he reported three surgical methods for the treatment of and, more important, the prevention of future attacks: — (a) Paracentesis, (b) Puncture and extraction of some vitreous, (c) Iridencleisis.

In 36 cases where hypopyon existed, paracentesis was carried out with the following results: — 16 recovered, 14 relapsed and in 6 cases follow-up was not possible.

Puncture of the vitreous was performed when the vitreous body was clouded with exudate. The purpose was to partially withdraw and thus change parts of the vitreous, and to substitute the removed portion with an equal amount of saline-penicillin solution. Of 11 cases treated, in 3, the vitreous cleared on the 3rd day and remained clear after one month; in 8, the vitreous remained as cloudy as before the operation, (in 1 of these 8, atrophy of the eyeball resulted).

Iridencleisis (incarceration of a pillar of iris under the bulbar conjunctiva) is indicated in those cases in which the iris is not atrophied to such an extent that it would tear when handled.

Out of 34 cases, 20 were operated on in the stage when there was no atrophy of the iris or cataract of the lens. In this group of 20, there were no relapses and some were under observation for as long as 5 years. In 3 of these the other eye became affected after some time.

Dimic goes on to state, "In our country, relapses of periodic ophthalmia in horses usually occur at intervals of two months. In this group of 20 operated animals, sight remained at least in hypofunction. The degree of hypofunction depended on the pathological and anatomical changes caused by the first attack. When we consider that medically treated cases of periodic ophthalmia usually have complete loss of sight within a year, the iridencleisis operation is clearly indicated in the treatment of this disease."

In the other 14 cases treated in this manner, two had severe inflammation of the cornea which remained cloudy. The remaining 12 were operated in the later stages of the disease in order to widen the pupil, but the results were negative. The iris tore and cataract developed.

Dimic then concludes: "Considering the possibility that periodic ophthalmia is an allergic disease, this allergen might be somehow connected with the pigment of the iris, which is produced in the form of synechiae and resensitises the eye which is already sensitised by the factors which caused the original attack. This explanation, in our case, would be similar to the theory according to which the pigment produced is an allergen. Sympathetic ophthalmia in humans is similarly interpreted by certain authors.

Our operation introduced this pigment under the conjunctiva and so brought about a desensitisation. But we must not lose sight of the fact that in the course of these immunobiological reactions, proteins play a certain role and these proteins cannot be separated from the other parts of the tissue."

"On the other hand this effect of the incarceration of the iris in the case of periodic ophthalmia might be explained by the effect attributed to this operation in cases of primary glaucomatous conditions. The traumatic action which irritates the front parts of the uvea causes changes in the neurovascular system. These changes cause vasodilatation and hypotonia. Even these changes might have a favourable therapeutic effect in the pathogenesis of periodic ophthalmia in horses."

Thus, it would seem that in view of the obscure etiology and the failure on the part of medical treatment to prevent recurrent attacks, surgical intervention at the first attack -or- at least no later than the second attack, is clearly indicated on the basis of Dimic's work.

DR. C. H. REID: We are finding more and more horses with leptospirosis, that do not have periodic ophthalmia. I want to leave that point but, having taken Filaria out of the anterior chamber of the horse many times, I think Roberts is on the right track, because Onchocera volvulus has been found in the front leg of the horse, in the tendons.

I believe that we don't do enough necropsies. When we do, we sometimes are surprised at the number of horses that have Filaria equina in the abdomen. Has there been any work done to try to correlate the presence of Filaria in the anterior chamber of the eye with the presence of Filaria equina elsewhere?

DR. MAGRANE: I don't know of any such work. You brought up a point I tried to re-emphasize, that, regardless of species, this is a multiple factor disease, that Leptospira definitely could be a causative factor, Brucella could, also Streptococcus equi, and on down the line. There is no one single factor involved; iridocyclitis in man or beast is the result of some systemic focus of infection, or some allergic response or hypersensitivity on the part of the eye. It is going to be extremely difficult, if not impossible, in many, many cases to ever determine the etiological factor. They are working on it all the time in the human. They are coming up with more and more toxoplasmosis in the human as a possible etiological factor of this same disease. In essence, we are just treating symptoms because of the lack of knowledge of the etiology in many cases.

QUESTION: Did Dimic wait until the second attack to make sure he was dealing with periodic ophthalmia?

XUM

DR. MAGRANE: Apparently not. I gathered from his paper that those in which he had 100 per cent results were one of two things; he couldn't quite be sure during the first attack, or he operated during the second attack when there had been no damage done in the first attack. He found there need not be any damage to the iris. I gather from his paper that it could be operated on in the second attack, provided the first attack was handled properly early, and there was no damage to the iris. In fact, I would be more inclined to go along with operating in the second attack. There you have the proof of the pudding, whereas you might be a bit dubious as to whether or not the animal is going to have a second attack, and you would be less inclined to operate at the first. The important thing was, of course, that there was no damage to the iris, in particular, when he operated. If he could operate in the throes of an attack, that was O.K., but he didn't want any weakening or atrophy present in the iris; otherwise his results were quite negative.

DR. REID: You get enough cases with trauma of some kind that you would be a little reluctant to operate.

DR. MAGRANE: There is the question of diagnosis in the initial stage. I am sure, and I am sure you will agree with me, there are many diagnoses of periodic ophthalmia in this country that are not really cases of periodic ophthalmia. I am certain of that because I have been called in on some cases which I was sure were not periodic ophthalmia but they were thought to be so. Every lesion of a horse's eye is not periodic ophthalmia. I again stress the importance of adequate examination in a semi-darkened area, with proper equipment. You can look beyond the cornea and see what is going on regarding the pupil of the eye and the anterior chamber, and so on. Then, when you put the findings together, you will be far more apt to make a diagnosis either of this disease or discount its presence.

QUESTION: Do you believe Vitamin A has any therapeutic effect on, or is a deficiency of Vitamin A a predisposing factor of eye conditions? I would also like to hear your opinion of the use of foreign protein which has been used in the past.

DR. MAGRANE: Vitamin A would have little or no effect on the course or prevention of this disease that I know of. As far as foreign protein is concerned, we used to use foreign proteins in cases of iritis and iridocyclitis. We have gone over to the use of corticoids. There is nothing wrong with the use of foreign proteins. I think the newer corticoids have taken their place.

QUESTION: After the incarceration operation, what percentage of vision — if any — do you figure is disturbed as a result of surgery?

DR. MAGRANE: You have reference to the dog?

QUESTION: Yes, in the dog.

DR. MAGRANE: If an eye that still has, let's say, 50 per cent vision, is operated by means of iridencleisis, in most instances the vision that is there at the time of surgery will remain, provided the operation is a success and that the tension goes back to normal and remains so. Although over the course of some months, and even years, as a result of the glaucoma, and even in spite of the fact that it was handled very early by surgery, certain lens changes take place. In other words, there may be cataract formation which would then blind the animal. Or, in other cases, the retina will undergo degeneration due to unknown causes. This is also true in many cases in the human where tension has been controlled, nevertheless blindness continues to be a slow, progressive sort of thing.

The trouble in the dog, with this particular operation is that you usually get them when they are blind, so the purpose of the surgery is more often to save the globe from the standpoint of cosmetic purposes than it is to save vision, although if it is treated early enough, the vision can be retained for many years.

QUESTION: Do you feel there is any benefit to incorporating penicillin-streptomycin with the cortisone?

DR. MAGRANE: In certain conditions, yes. I am not sure in this particular condition, since it is an involvement of the uveal tract. Again, it is coming from somewhere in the body, and it is treated best systemically or by subconjunctival injection. Subconjunctival injections of antibiotics such as neomycin or chloromycetin can be used, but we usually use them in acute ulcerative conditions and where we have infection of the corneal conjunctiva. They can be incorporated, of course, with corticoids. In those conditions (ulceration) we are using them alone.

It has not been very popular to use subconjunctival antibiotics in veterinary and human medicine. They can be used if the dilution is correct. This is important because necrosis may occur at the site of injection or underneath the conjunctiva, if used in too high a concentration. This has all been worked out in the human.

QUESTION: Do you use any chymar?

DR. MAGRANE: We have used chymar on the dog. We think it is extremely helpful. In cases where exudate has failed to drain properly from the anterior chamber in a valuable animal, we use chymar, morning and night for a period of four or five days. We think it definitely has helped to absorb some of the exudate in the anterior chamber. It is an extremely expensive preparation, as you will know, if used over a long period, or in large doses.

XUM

QUESTION: I get the impression you favor this operation perhaps in a horse with the disease. Have you yourself done enough of this surgery in the horse to say that there would be no side-effects, and so forth?

DR. MAGRANE: From Dimic's work, I am fairly well convinced that this operation has a place. I have done it only experimentally, and that was some years ago. I do it all the time in the dog's eye. The operation in the horse is relatively easy. Once you obtain anesthesia and control the nystagmus, the horse's eye is far easier to operate on than the dog's. In the first place, you have a larger globe; secondly, the structures are actually softer and not as tough as in the ordinary dog's eye. The operation is relatively simple, once you understand the procedure. As far as results are concerned we have only Dimic's work to go by.

PHYSICAL EXAMINATION OF THE HORSE FOR SOUNDNESS

JOHN E. PETERS

To conduct an examination for soundness in a horse, let us consider a hypothetical case — that one of us has been sent to a sale by a client who is in the market for a racehorse; or we have been asked to go to a race track to examine a particular horse in training. In either case, the examination should be conducted to the best of one's ability and the animal passed or rejected accordingly. The client's reason for wishing to purchase the horse will determine if a certain unsoundness is acceptable. For example, the animal in question is a mare which has raced hard, has a good racing record but has developed a bowed tendon. Knowing that our client plans to retire this mare to stud, we would surely not turn her down because of this condition. From this, it is evident that soundness in a horse is relative to the use for which the animal is intended.

At this time we shall consider the physical examination for soundness only from the racing point of view.

If any of us were attending the same sale and could see the same good qualities in an individual, we would probably want the same horse. This, however, is not usually the case. My reasons for liking the conformation and soundness of a particular horse may not coincide with yours. I firmly believe that, based on looks, conformation or soundness, one cannot say that one horse will run faster or farther than another. Hard though we may try to sort out a few good individuals from a group, we fall far short of being one hundred per cent correct.

Runners come in all sizes and shapes as you are all aware from watching our racehorses perform at the various tracks. Personally, I would much rather have a good, sound, little horse than a good, sound, big horse. The smaller horse will have a much better chance of remaining sound than the larger animal which carries more weight and always hits the ground hard. This is not to say that there are no good, big horses.

Looking at winners of races, we seldom find an individual which, after constant training and racing, remains completely sound in all respects. If we do, it is the exception rather than the rule. A common saying is "If a horse is sound, he cannot run fast enough to break down!"

Other factors which play important roles in the matter of

XUM

soundness are proper conditioning, correct treatment and scientific nutrition, to mention only a few.

Now, for the hypothetical case, let us go back and determine by a more thorough examination if this horse, by its conformation and soundness, has the potential qualities of a superior racehorse. Obviously, our selection would be influenced by good breeding but our topic eliminates the discussion of these factors.

When we first see the horse, we shall be either impressed or unimpressed. If the latter, there must be somthing undesirable in its conformation or condition. It may be size, crooked legs or some obvious unsoundness; but if the horse appeals to the examiner, a closer inspection will be indicated to eliminate any obscure, undesirable qualities.

The forelegs are of the utmost importance; first, because they carry more than half of the total weight of the animal. We have all seen pictures of the running horse where all the weight is thrown on one front leg: second, because unsoundness occurs most commonly in the forelegs, although there might be some question about the importance of this in trotters and pacers.

The hind legs are often seen to be rested by shifting the weight from one to the other, but each front leg continuously bears equal weight. The fact that the front legs are never at rest while the hind ones sometimes are, means that the stay apparatus is better developed in front. If an animal should rest or point a front leg, it is certain that pain is present.

With the conclusion of the general impression, attention should be directed to the foot and pastern. To quote another old saying — "no foot, no horse". First, examine the general shape and angle of the hoof, the size of the hoof as it is growing in relation to the pastern. Check that the hoof is not dished, for a dished hoof always indicates an abnormal growth of the wall. This is usually due to injuries to the coronet or the presence of ringbone. Check the wall for the presence of any tell-tale grooves, such as founder grooves or quarter crack grooves. The surface of the sole should be concave, the frog well-formed and the heels neither too high nor too low. The area of the coronary band should be closely examined for injuries, scars and evidence of sidebone development.

The pastern should be short with a good slope towards the fetlock. A pastern, too straight and short, would obviously offer little spring and absorb less shock. In pasterns that are too straight, a greater incidence of ringbone is noted, but on the other hand, a pastern that is too long and sloping, increases the strain on the flexor tendons. The ideal then, is a medium length pastern with an average slope towards the fetlock.

The length of the cannon bone in relation to the limb is considered. A short cannon bone is desirable, for the shorter the bone, the less strain will be imposed on the flexor tendons. Invariably, with a short, flat cannon, will be found well-developed tendons and ligaments which are so necessary for the absorption of extreme shock and pressure when a horse is in full stride. Abnormalities of the splint bones should be noted at this stage.

The suspensory ligament is very important because it takes the weight of the horse at each stride and is the main structure which prevents the fetlock being forced into extreme extension. A good test to apply to any horse is to pick up the leg and with the thumb and forefinger apply some pressure to the suspensory ligament from the region of the ankle to a point above the bifurcation. In an abnormal ligament, some degree of sensitivity will be noted. The suspensory ligament will be one of the first structures involved in an unbalanced horse with poor tracking ability. When weight is taken on the leg, no tenderness will be noted on digital pressure unless an advanced degree of tendonitis is present. I am excluding, of course, tendonitis due to injuries or blows to the area. The leg should always be lifted when examining the tendons.

Observing the foreleg from the side, an imaginary line should fall straight from the middle of the forearm and pass through the knee and cannon bone to the fetlock. If the horse is a little over-at-the-knee, it is a lesser fault than the opposite condition of back-at-the-knee or calf-kneed. If the knee is sprung backward, to the inside or the outside, the gait is apt to be ungainly with undue strain being placed on the tendons, leading perhaps to an early breakdown.

As the examiner faces the horse, there should be good width between the two front legs as they "emerge from the trunk". An imaginary line through the forearm, knee, cannon, fetlock and hoof will indicate any portion of the limb that is offset. If such offset involves the cannon region, it will usually be on the lateral part of the knee joint. This is of significance since it occurs at one of the main points of concussion. Unless the knee is of good width, and the cannon bone directly in line with the forearm, sooner or later the development of carpitis or splints or both may be anticipated. If the area involved is from the fetlock to the hoof, the offset may be medial or lateral. This indicates that the horse is toed-in or toed-out.

With regard to the hind leg, (starting at the hoof and working up to the hock), the examination should be carried out in a similar fashion to that of the front leg.

In the hock, one should look for the presence of bone or bog spavin and at the back of that joint and below the tuber calcis,

XUM

curb should be looked for. Curbs may be of little importance unless large and in association with an exaggerated angulation of the hock. On the medial aspect, at the seat of spavin, examination should be aimed at the detection of cuts, scars or other injuries which might be evidence of earlier speedy cutting.

In the thoroughbred, it is very seldom that one finds ligament or tendon trouble between the hock and fetlock and only occasionally are splints noted in the hind leg.

A hock which is bent inwards from the imaginary line drawn down the back of the leg and passing through the hock and fetlock, lends itself to many chances for injury. Due to improper alignment, concussion may be so concentrated that bone spavin may be an early development.

Following the examination of the horse at rest, attention is paid to the animal at the walk. It is important that the examiner stands in front of the horse as it walks towards him, so that the manner of picking up and bringing forward the feet may be observed. The feet and legs should be advanced in a straight line without any inward swing or paddling action. A normal straightforward action seldom will give rise to brushing, scalping or speedy cutting.

If the animal in question is unbroken it should be turned out in a paddock where his movements and action can be clearly seen. If the horse is in training, it should be saddled and taken to the track and examined at the gallop. One should look for any wrong steps the horse takes after the boy has been placed on its back. If possible he should be followed to the track and his actions observed. Note should be made of the placing of the feet with reference to each other. Many good horsemen have said "The farther he oversteps with the hind foot, the better the horse he is". There may be some merit in this statement.

Next, the horse should be examined as it warms up. View the horse as it comes towards you at the slow gallop, then step behind as it goes away from you. This should be done also as the horse is breezed. Notice should be taken of the gait, action and rhythm and whether the horse weaves or travels in a straight line. As it breezes by, there will be an opportunity to listen to its breathing. Sale horses are guaranteed sound in wind and eye unless otherwise stated.

If after this complete physical examination, you are sure that this horse is perfectly sound, or as nearly sound as he need be for the intended purpose, you will very likely purchase him for your client, hoping he will be a great horse.

There is never a guarantee that this animal will stand through

months or years of training and racing, but I believe you can be assured, barring accidents, that he will have a more than equal chance.

In closing—we all know that soundness is perhaps the first requisite for a good race horse, but there are several others. We can never underestimate the need for unquestionable pedigree.

In spite of these specific, scientific, imperative requirements, we know that there is that abstract, difficult-to-describe quality—call it sixth sense, intuition, hunch—or just plain luck, which encourages one of us to want a certain horse in preference to another—why?—who knows?

All the scientific knowledge available is still woefully inadequate for the demands sometimes made on veterinarians. There are certainly no pat answers for all our questions. We often must rely on our own art—which is our own individual ability, to draw upon our past experiences, skills, knowledge, intuition and apply them to our everyday needs.

I recently heard the definition of a scientist which seems to apply to the veterinarian:

"One who has the simplicity to wonder
The ability to question
The power to generalize
And the capacity to apply".

XUM

NUTRITIONAL DISEASES OF DOGS AND THEIR POSSIBLE RELATIONSHIP TO THE HORSE

WAYNE O. KESTER

I should like to open this discussion with a quotation from a recent A.V.M.A. Journal. It is entitled "EQUINE NUTRITIONAL FACTS".

> "Few specific nutrient requirements of horses have been established through controlled research. Using information available from other species with due regard for anatomic and physiologic differences, however, horses can be fed well-balanced rations composed primarily of naturally occurring feedstuffs. Such feeding programs may have to be supplemented with minerals and vitamins. Until more controlled experimental work is conducted and quantitative requirements for various nutrients determined, we shall have to rely to a great extent upon practical experience and the correlation of information derived from other species. —"

During the next few minutes we will review some research work done in another species — the dog. It has to do with a skeletal disease problem observed and studied in the Great Dane. At the moment no direct connection between it and skeletal disease in the horse is definite, yet there are so many striking similarities in history, clinical symptoms and pathology, that it gives one cause for wonder.

In addition, some conclusions were drawn regarding the hazards of using the various minerals excessively as feed supplements, which we have every reason to believe are as applicable to the horse as they are to the dog.

First, a brief history of the case will be presented; second, the research and conclusions drawn from it; and third, some slides and movies to further illustrate the clinical symptoms and lesions.

CLINICAL HISTORY OF THE INVESTIGATION:

A Great Dane bitch with a litter of eight pups was brought to the attention of a practitioner. One pup was lame. All pups had enlarged, knobby carpi; radiographs indicated that all had bone abnormalities. Massive doses of vitamin D, calcium and phosphorus were then administered. Within a few days clinical symptoms indicated the condition was rapidly getting worse.

XUM

A review of feeding practices indicated that the bitch had been on a ration inadequate to provide total energy and protein requirements during the gestation and lactation period. Protein requirements in the pups had probably been deficient for some time and the developing skeletal condition was believed to be irreversible.

At this point the bitch was purchased and housed at the Morris Research Laboratory Farm where she subsequently, under various feeding regimes, produced four litters of pups by different sires. Clinically, the bitch appeared to be normal throughout her lifetime. An apparently normal daughter of this bitch also produced one litter. These five litters, making a total of 36 dogs, were involved in this study.

Litter #1: Prior to breeding, the bitch was placed on a good quality basal reproduction diet supplemented with fresh meat and liver. This ration was believed to have adequate amounts of calcium, phosphorus and vitamin D, also digestible sources of energy, including fats. This diet had already been thoroughly tested in the Beagle and other small dogs and found to be entirely adequate in that all test dogs remained symptom-free.

Clinically, this litter appeared normal until five months old. At this time, which was the high point of skeletal stress during this growth period, the pups contracted an encephalitis-distemper-like virus infection and 4 of the 7 died. Only a very few of the other 100 dogs in the colony developed symptoms of the virus infection, none of them severe. No treatments were administered during the outbreak and no vaccinations of any kind are ever used. This colony is, however, handled exceptionally well from a management standpoint. Housing and exercise facilities approach as nearly as possible the natural environment for the dog and all dogs are maintained on what is believed to be the best possible nutritional balance. This experience indicates that such an environment is conducive to a markedly lower morbidity and mortality rate.

Interestingly, the virus infection manifested itself in this litter during the height of the growth stress period. Subsequent radiographs and autopsies disclosed that this litter had subclinical involvements with the skeletal disease in question. Obviously, we were dealing with a complicated metabolic problem.

Litter #2: For this litter, the same diet was used on the bitch and pups except that the calcium level was increased. Analytical data indicated adequate phosphorus but that the calcium level may have been slightly low. Clinical symptoms of the skeletal disease began to appear and at three months of age, increased vitamin D was given to two of the pups. The pup on the highest level of calcium promptly developed marked leg lesions.

It was concluded that the disease followed a definite pattern and was not reversible. Massive doses of vitamin D alone were not helpful, in fact, were actually detrimental. The value of increasing calcium alone was questioned.

Litter #3: This litter was handled essentially the same except that calcium and vitamin D were used in increased amounts. This litter also manifested symptoms of skeletal disease and it was further concluded that increasing the calcium and vitamin D levels was not effective in preventing this disease. The adequacy of the available phosphorus level was still in question.

Litter #4: The bitch and litter were placed on a different diet but analytically similar to previous diets except that the calcium, phosphorus and vitamin D levels were lowered comparable to that used on the first litter. Due to processing methods, however, these components were believed to be biologically more available than in previous diets. This litter showed no clinical manifestation of the disease. A series of radiographs indicated there had been no skeletal involvement typical of the disease in question.

Litter #5: This litter was whelped by an apparently normal daughter of the original bitch. The same basal diet was used as with the first three litters except that it was fortified with calcium, phosphorus and extra vitamin D. This litter showed no clinical symptoms; however, radiographically and at necropsy, lesions were identified on some of the pups.

PICTURES AND ILLUSTRATIONS:

The first three slides will show some of the typical clinical symptoms: crooked legs, enlarged bones and joints, bone curvature, splay feet, spread and flattened toes and pads.

The next series are selected radiographs which depict the disease as seen by the radiologist, Dr. Carlson of Colorado State University, who will describe these plates.

DR. CARLSON:

[Slide] The first slide is typical of classical rickets. There is an increased density at the metaphysis of the long bones, with some marginal lipping and increased width, which is manifest in both the anterior and posterior view. In addition, this particular animal had curvature of the associated radius and ulna.

[Slide] This slide presents another animal showing these changes. Again, the increased width, the marginal lipping just above the epiphysis and the increased density. You can also see the curvature of these bones. It is my opinion, and I am sure a number of people agree with me, that these changes occur in most large breeds of dogs in their developmental period, and I seriously question if this does represent a true case of rickets even though the changes here are those described under "classical" rickets.

XUM

We need considerably more work in this field to actually determine what is going on. I do question if there is any deficiency of calcium and associated deficiency, such as rickets. I am sure General Kester and Mark Morris agree with me on some of these opinions. This is not the typical picture of rickets as seen in humans.

[Slide] This is the case in which there had been excess vitamin D supplement. In the periarticular tissues, in the joint capsules and in the bursa, are seen multiple masses of calcified densities or increased densities. This is an extra-skeletal deposit of calcium in the peritendinous tissues and the bursa surrounding the joint. This is not ossification; it is true calcification.

In this area there is some curvature, also some decrease in density, which most likely represents a decalcification due most likely to this condition. This is a typical textbook description of hypervitaminosis.

The implications that can possibly be drawn from this will be discussed in a few moments by General Kester.

DR. KESTER:

It was concluded from the foregoing that the condition was probably not inherited. The disease appears to be a multiple nutritional deficiency created partially by the rapid growth of the animal resulting in marginal deficiencies of certain limiting dietary constituents. It is probable that a part of this condition is initiated in utero by a nutritional deficiency. Once the syndrome is apparent we know of nothing that will alleviate it. Increased dosage of vitamins and minerals seems, in fact, to worsen the condition by further disturbing the nutrient balance.

OBSERVATIONS FOR CONSIDERATION:

In the embryo, the skeletal matrix derived from protein is first laid down before mineralization takes place. Bone is built from protein or rather, amino acids derived from proteins which must be in proper ratio. Consequently, biologically available proteins of the proper type must be supplied in adequate amounts during pregnancy, lactation and the growth cycles. Minerals and vitamins may not be properly utilized in the absence of these essential proteins. Vitamin-mineral supplements to a ration deficient in available proteins appear to be of little value, in fact, in some instances are detrimental because they further upset an already precariously balanced metabolism.

The reserve pools of protein and energy in the dam must be filled prior to and during gestation if proper skeletal formation in the embryo and growing offspring is to take place.

There was an unexpectedly high requirement for food during the lactating and growth cycles. On the fourth week of lactation

the Dane bitch consumed 20-25 pounds of moist food (66% moisture) per day. The need is for total balanced energy each day — not supplements.

Do we recognize a comparable requirement in the mare?

The stage for this skeletal disease appears to be set in utero. However, symptoms do not appear until the most rapid growth period, about the 12th to 16th week of life, and then only if the diet is inadequate.

Do we have a similar situation in the equine?

This condition has been reported in other large dogs; however, so far, we have seen it only in the Dane. Is this because he is a Dane or because the Dane has an unusually rapid growth period? All pups in this study were fed well and pushed for growth.

Again, do we have a similar situation in the rapidly growing colt as produced for today's market?

After the rapid growth period the deficiencies are rapidly made up, followed by over-compensations and tissue distortions resulting in abnormal new bone proliferations.

Do we have a parallel in the overfed rapidly growing young equine?

Interestingly, these pups seemed most susceptible to infectious disease during the height of stress in their rapid growth period and more susceptible than pups which had reached a less rapid growth period.

Again, is there such a cycle in the horse?

SUMMARY:

What does all this mean in relation to equine medicine? Mostly it opens a box of unanswered questions for us to ponder and hope someone in research will soon step forth with the answers.

It rather confirms, for me, a long-standing belief that the promiscuous over-feeding of vitamins and minerals especially to mares and foals is a hazardous procedure, also that the empirical therapeutic administration of vitamins and minerals will give spectacular results both good and bad.

Satisfying protein requirements with respect to type, quality and availability takes on new importance, especially in the brood mare and foal. Perhaps in our problem cases we should first check the ration as to quality, quantity and availability of digestible proteins, fats and other energy sources before we worry too much about vitamins, minerals, tonics, parasites, teeth and other factors related to digestion and nutrition.

XUM

SOME DRUGS AND THERAPY AFFECTING THE GASTRO-INTESTINAL TRACT

S. F. Scheidy and J. E. Martin

(Presented by the Senior Author)

The two groups of drugs commonly employed to alter motor and secretory activity of the gastro-intestinal tract are those that exert their effects on the parasympathetic nervous system, either stimulating or inhibiting its activity. Although the gastro-intestinal muscle possesses an intrinsic activity of its own, the parasympathetic nervous system plays a major role in increasing or decreasing the tonus and peristaltic motility of the muscle as well as the rate of flow of many secretions.

Drugs which inhibit parasympathetic activity are commonly used to treat diarrhea and to overcome spasm of the bowel. They also are used to reduce the flow of acid gastric juice in treating ulcers and other gastric disorders in man and, to some extent, in dogs. Drugs which stimulate the parasympathetic nervous system are used less extensively, and then mainly as cathartics or in treating certain types of colic in horses.

While both groups of drugs are used in veterinary practice, mainly for their effects on the gastro-intestinal tract, it is important to recognize that the drugs act on the parasympathetic nervous system, in general, and can produce other effects in addition to those exerted on the alimentary tract. These other effects or side-effects may be undesirable and in fact, in certain instances can be dangerous. Therefore, in using these drugs the overall condition of the patient must always be carefully evaluated in relation to parasympathetic activity.

Clinical problems affecting the gastro-intestinal tract in horses are chiefly infections due to micro-organisms and parasites, as well as the recognized functional disturbance in the digestive process generally referred to as "colic". The infections due to micro-organisms can be controlled usually by the use of sulfonamides and antibiotic agents. Foal scours are favorably influenced by sulfaguanidine[1] and phthalylsulfathiazole[2].

In some animals, systemic infections also occur and are combatted by agents having a wide range of antibacterial activity and which are well distributed throughout the body. Thus, compounds that have systemic as well as anthelmintic actions are used to overcome certain infections.

XUM

In addition to the above, there is an indication that a combination or concomitant therapy with chemotherapeutic and pharmacodynamic agents is useful in the management of certain intestinal disturbances, especially those accompanied by chronic diarrhea.

The purpose of this paper is to review some of the major actions of drugs affecting the parasympathetic nervous system and in particular, to discuss briefly their effects in the horse. Actually, although these drugs are used clinically for certain purposes in horses, there have been few reports of their overall physiological effects in this species.

PARASYMPATHETIC-STIMULATING AGENTS:

These drugs act directly on the effector cells, producing an effect similar to that observed when the parasympathetic nervous system is stimulated electrically. The parasympathomimetic drugs are of two general types:

1. Choline esters, which closely resemble acetylcholine, the chemical agent responsible for transmission of impulses from parasympathetic nerve endings to visceral muscle. Examples are carbachol and bethanechol (Urecholine.(R))*
2. Quaternary compounds, not directly related to acetylcholine but possessing many of its actions. Other compounds such as arecoline and pilocarpine produce a similar action.

The cholinesterase inhibitors produce a parasympathetic effect by preventing rapid destruction of the chemical mediator (acetylcholine) responsible for transmission of impulses at the neuromuscular junction. This is brought about by their inhibition of cholinesterase, an enzyme which normally destroys acetylcholine at a rapid rate and prevents its accumulation at the neuromuscular junction. Examples of cholinesterase inhibitors are physostigmine, neostigmine and many of the organic phosphate insecticides.

CARBACHOL

GENERAL ACTIONS:

Carbachol (Lentin®)** is a parasympathomimetic drug and as such exerts the following general actions:

1. Increase in pulse rate.
2. General vasodilatation with a lowering of blood pressure.
3. Increased secretion of sweat, bronchial mucus and saliva.

*Urecholine (brand of bethanechol) is a product of Merck & Co., Inc., Rahway, N. J.

**Lentin (brand of Carbachol) is a product of Merck & Co., Rahway, N. J.

4. Increased peristalsis of intestinal, uterine and urinary bladder musculature.

5. Contraction of the pupil.

ACTIONS IN THE HORSE:

Although carbachol has been used frequently for clinical purposes in the horse there do not appear to be any published reports of its actions in this species. In limited studies on horses in which carbachol was administered subcutaneously in doses of 2 or 3 mg the following have been observed[3]:

1. Within 10 minutes of injection, the pulse rate increases and remains above the control value for 40 to 50 minutes. An average increase of 16 beats per minute was observed. Although only a few blood pressure determinations have been made, it is believed that the increase in pulse rate may be a compensatory reaction to a decrease in blood pressure. Another contributory cause of the rapid pulse might be the apparent colicky pain exhibited by some horses. In any event, despite the fact that carbachol would be expected to directly slow the pulse rate, on account of vagal stimulation, nearly all horses respond with an increased rate.

2. Increased salivation is observed within three to five minutes of drug administration, and by 10 minutes the flow is quite copious and watery. This action lasts for 60 to 90 minutes.

3. Peristaltic activity of the intestine (as judged by auscultation) increases within five to ten minutes. The intestine remains more active for periods up to 60 minutes or longer. Horses usually defecate one or more times during the first 15 minutes after carbachol injection. One horse defecated 12 times in a one hour period. The stools tend to become softer and the horse may show restlessness and evidence of a mild colic. Flatulence is commonly observed.

4. Some, but not all horses sweat after injection with carbachol. This, however, is usually not evident until about one hour after administration of the drug.

5. A rather constant occurrence following carbachol medication is the appearance of a watery, nasal discharge. This commences within 10 minutes and may last for up to one hour.

PHYSOSTIGMINE SALICYLATE

Physostigmine salicylate is the salt of an alkaloid obtained from seeds of **Physostigma venenosum**, sometimes referred to as the calabar bean plant. Physostigmine salicylate is also referred to as eserine salicylate.

XUM

GENERAL ACTIONS:

Physostigmine exerts its stimulant effect on the parasympathetic nervous system by combining with cholinesterase and preventing this enzyme from inactivating acetylcholine. Thus, acetylcholine is able to accumulate and have a prolonged action on the cholinergic effector cells.

The important effects of therapeutic doses of physostigmine are:

1. Increased motility of smooth muscle of the gastro-intestinal tract and some increase in secretions of the digestive tract. However, the latter effect is not so marked as that observed with carbachol and pilocarpine.
2. Constriction of the pupil, either when applied topically or administered systemically.
3. Stimulation of skeletal muscle.
4. Slowing of the heart rate.
5. Contraction of smooth muscle of the bladder.

ACTIONS IN THE HORSE:

Only limited studies have been conducted on the pharmacological actions of physostigmine in the horse. The following have been observed when the drug is administered intravenously in a dose of 0.045 mg per pound of body weight: (45 mg/1000 lbs).

1. **Heart Rate**
   Initially, the heart rate shows a sharp increase of 10 - 20 beats per minute but after approximately 15 minutes it becomes slower than the control rate. This persists for up to six hours. The initial increase in heart rate may be a compensatory effect for peripheral vasodilatation and subsequent lowering of blood pressure. However, at the moment there are no reported studies of the effect of physostigmine on blood pressure in the horse. The overall effect of physostigmine on heart rate in the horse differs from that of carbachol which causes an increased rate without a subsequent slowing.
2. **Respiratory Rate**
   Within five minutes of injection of physostigmine there is a sharp increase in the respiratory rate. In some horses, the rate may increase to twice the control value. The increased rate may persist for as long as six hours. This effect has not been observed with carbachol.
3. **Salivation**
   An increased flow of saliva begins within five minutes of injection of physostigmine. The saliva produced is thin and watery.

4. **Perspiration**

   Most horses sweat within the first 30 minutes after physostigmine administration. This effect lasts for approximately two hours.

5. **Other Effects**

   Some horses show moderate excitement following intravenous injection of physostigmine in a dose of 0.045 mg per pound of body weight (45 mg/1000 lb).

   There is repeated defecation during the first 30 minutes. The stools become loose and there is frequent passage of flatus.

6. **Antagonistic Effects of Atropine**

   In doses of 0.009 to 0.02 mg per pound of body weight (9 to 20 mg/1000 lb), atropine will block the effects of physostigmine (0.045 mg/pound intravenously) on the respiratory rate and will depress its effects on salivation, defecation and sweating. However, in these doses, atropine did not block the effect of physostigmine on heart rate.

OTHER DRUGS THAT STIMULATE THE PARASYMPATHETIC NERVOUS SYSTEM:

There are a number of other drugs which stimulate the parasympathetic nervous system but they have not been studied in the horse, and therefore, are not in common use in this species. At one time, arecoline hydrobromide was used in horses as a rapidly acting cathartic, however, it has now been replaced for this purpose by safer drugs. Another drug which stimulates parasympathetic activity is pilocarpine. Both arecoline and pilocarpine stimulate effector cells directly and share the general actions described for carbachol. Pilocarpine is noted for its potent action in stimulating salivation and gastro-intestinal secretions.

Neostigmine bromide, also referred to as prostigmine bromide, is a cholinesterase inhibitor. Its effects are similar to those of physostigmine but it is somewhat less potent.

PARASYMPATHETIC BLOCKING AGENTS:

These drugs inhibit the transmission of impulses from parasympathetic nerve endings to visceral muscle. In a sense they prevent acetylcholine, liberated from a parasympathetic nerve ending, from activating the tissue which it innervates. They are also referred to as anticholinergic agents, cholinergic blocking agents and parasympatholytic agents. Common examples of drugs in this group are naturally-occurring belladonna alkaloids, (e.g. atropine, scopolamine) and a large group of synthetic compounds such as homatropine, isopropamide, methantheline and diphenmanil.

XUM

ATROPINE SULFATE

GENERAL ACTIONS:

Atropine, the principal alkaloid of belladonna, is capable of preventing the interaction of acetylcholine with the effector cells.

The exact mode of this action is not known. The principal effects of therapeutic doses are[4]:

1. Acceleration of heart rate, and some increase in blood pressure.
2. Dilatation of bronchioles and drying of secretions of the respiratory tract.
3. Dilatation of the pupil.
4. Inhibition of secretion of saliva and gastric juice.
5. Decrease in motility and tonus of gastro-intestinal musculature.

ACTIONS IN THE HORSE:

The effects of atropine in varying doses on certain functions in the horse are as follows:

1. **Cardiac Activity:**
   The heart rate in the horse is quite sensitive to the action of atropine. In doses as small as 0.009 mg per pound of body weight (i.e. a total dose of 9.0 mg for 1000 pound horse) there is a perceptible increase in heart rate. With increasing dosage, the heart beats more rapidly so that with a dose of 0.11 mg per pound, rates as high as 104 per minute are observed. The electrocardiogram exhibits some changes in the form of shortened PR, QRS and QT intervals. In some instances a transient, partial heart block occurs at the time when the heart rate is accelerating. This is possibly explained by the fact that atropine is known to cause a transient increase in vagal tone before peripheral blocking of vagal activity occurs.

2. **Blood Pressure and Respiration:**
   In doses ranging from 0.009 to 0.11 mg per pound, atropine does not cause any consistent change in blood pressure or in the respiratory rate or rhythm in the horse.

3. **Pupil:**
   With doses above 0.02 mg per pound, atropine produces mydriasis which may last up to five hours.

4. **Gastro-intestinal Motility:**
   With doses above 0.02 mg per pound, gastro-intestinal mo-

tility is depressed by atropine. The effect may begin in as little as five minutes after subcutaneous injection of the drug and may last as long as four hours. In doses of 0.05 mg per pound, atropine is capable of blocking the stimulant effect of physostigmine (0.04 mg per pound).

5. **Behavior:**
With subcutaneous doses of 0.05 to 0.1 mg per pound, horses consistently show marked excitement. This takes the form of apparent hallucinations, muscle tremors, frequent urination and inco-ordination. The effect starts in about 30 minutes and may last for several hours. Smaller doses (0.02 to 0.04 mg per pound) cause excitement in some horses but not in others. Doses lower than 0.02 mg per pound do not produce excitement.

ISOPROPAMIDE

Isopropamide iodide ('Darbid'(R))* is a synthetic anticholinergic drug. Chemically it is (3-carbamoyl-3,3-diphenylpropyl) diisopropyl-methylammonium iodide, and it is classified as a quaternary ammonium compound.

GENERAL EFFECTS:

Isopropamide produces the general effects characteristic of anticholinergic drugs including atropine. However, the intensity of certain of its effects and its length of action differ from those of atropine and other drugs of this group. In man, and in the dog, isopropamide inhibits gastric secretion and exerts a spasmolytic effect on intestinal musculature for as long as twelve hours after a single oral, therapeutic dose[5]. Some of the troublesome side effects observed with atropine (e.g. marked dryness of the mouth, blurred vision and difficult urination) are not usually encountered with isopropamide except when large doses are given.

ACTIONS IN THE HORSE:

Only limited studies have been conducted on the actions of isopropamide in horses[6]. In these studies, the primary objective was to determine the minimum dose of isopropamide which would exert certain actions and which would block the effects of cholinergic parasympathomimetic drugs. Therefore, the doses mentioned do not necessarily reflect those which would be of most value clinically. Further, in most instances the drug was used parenterally; in practice, oral administration might prove more desirable. When given to the horse in a single intravenous dose of 15 mg isopropamide exerts the following actions:

*Darbid is a product of Smith Kline & French Laboratories, Philadelphia, Pa.

XUM

1. Intestinal activity (as judged by auscultation) is inhibited almost immediately. This effect persists for three to four hours.
2. Salivation is inhibited within five minutes and although the mouth does not become excessively dry there is a noticeable decrease in moisture for approximately three hours.
3. Pulse rate and breathing rate are not significantly affected except that some horses may exhibit a slight increase in pulse rate.
4. No changes in behavior are observed with this dose.

In order to determine the possible blocking effect of isopropamide on the action of cholinergic drugs, it was given in conjunction with carbachol. In these studies isopropamide was administered intravenously (5 or 15 mg dose) either simultaneously with carbachol (subcutaneous injection of 2 or 3 mg) or within 20 to 30 minutes after carbachol injection. The following effects were observed:

1. Isopropamide, in the doses stated, does not consistently nor completely inhibit or overcome the increased pulse rate produced by carbachol in the horse.
2. In doses as low as 5 mg isopropamide will inhibit the excessive salivation ordinarily produced by carbachol. Further, if isopropamide is given after the salivary response has been allowed to develop it will inhibit salivation within two to five minutes.
3. The increased peristaltic activity and defecation observed with carbachol is almost completely inhibited by a 15 mg dose of isopropamide. The effect lasts for nearly two hours. A 5 mg dose of isopropamide is adequate in this respect.
4. In doses of 5 or 15 mg isopropamide inhibits the watery nasal discharge which usually accompanies the administration of carbachol.
5. Isopropamide does not inhibit the sweating sometimes observed in horses after injection of carbachol.

OTHER DRUGS WHICH INHIBIT THE PARASYMPATHETIC NERVOUS SYSTEM:

There are a great number of other drugs which have been developed for purposes of blocking the activity of the parasympathetic nervous system. In most instances the emphasis has been on finding drugs which markedly depress gastro-intestinal motility and secretions but have less activity than atropine on other por-

tions of the parasympathetic system. These drugs are frequently employed in human medicine and to some extent in dogs and cats but only rarely in horses. There is a great need for information on their actions in this species.

Homatropine is a synthetic drug with actions closely resembling those of atropine. While it has less side-effects than atropine, homatropine is only about one-tenth as potent in controlling gastro-intestinal spasm.

Methantheline (Banthine®)† is an example of a newer parasympatholytic drug having a marked effect in depressing gastrointestinal motility and secretions but exerting a relatively mild action on other portions of the parasympathetic nervous system. It is frequently used in man for the management of peptic ulcer and in controlling hypermotility and spasm of the intestinal tract. Aminopromazine (Jenotone(R))*, a phenothiazine derivative, possesses musculotropic activity and apparently is effective as a spasmolytic agent. It has been used in the treatment of chronic diarrhea, apparently with resulting hypermotility of the intestinal tract. However, we are not acquainted with any specific studies conducted in horses.

Diphenmanil methylsulfate plus antibiotics (Diathal(R))** is used in man for the same purposes as methantheline and has been used in dogs as well as in horses and cattle for the control of diarrhea. However, no information is available regarding the pharmacologic effects in horses.

A combination of isopropamide and proclorperazine (a phenothiazine tranquilizer) designed for use in humans has also been beneficial in the management of chronic diarrhea in equine patients[7]. It would appear that this approach to a frequently encountered problem should be further investigated.

SUMMARY:

The drugs most commonly used to increase or decrease gastrointestinal motility and secretory activity are those which exert their effects through an action on the parasympathetic nervous system. While some of these drugs have been used clinically in horses, there is a definite need for more information on their overall effects in this species. This is particularly true of a number of drugs which block the activity of the parasympathetic system

†Banthine is a product of G. D. Searle & Co., Chicago, Ill.

*Jenotone is a product of Jensen-Salsbery Laboratories, Kansas City, Mo.

**Diathal is a product of Schering Corporation, Bloomfield, N. J.

XUM

and which would appear useful in the management of diarrhea and other conditions in which there is hypermotility of the intestinal tract.

REFERENCES

1. Maderious, W. E. and Traum, J.: Successful Treatment of Foal Scours, Cornell Vet. 37: (1947), 259.
2. Clarke, H. E.: Use of 'Sulfathalidine' (phthalylsulfathiazole) in Foals. Vet. Med. 44: (1949), 272.
3. Martin, J. E.: Unpublished data.
4. Detweiler, D. K. and Martin, J. E.: Unpublished data.
5. Grossman, M. I.: Gastroenterology 35: (1958), 312.
6. Martin, J. E.: Personal observations (1961).
7. Anderson, Wm.: Unpublished data.

QUESTION: I would like to ask about the use of A.C.T.H. in chronic diarrhea. It has worked sometimes in cows: the reason for its action being unknown. I was interested in the decreased eosinophil count when atropine is used — similar to that observed with A.C.T.H.

DR. SCHEIDY: We personally have no experience with it. Theoretically, we believe there are some situations where A.C.T.H. might be indicated as is the case with cortisone.

QUESTION: I have used it clinically and it works. What are the mechanics of its action?

DR. SCHEIDY: I am not sure I can answer that.

AFRICAN HORSESICKNESS

F. D. Maurer

EDITOR'S NOTE:

As mentioned in the foreword, it was not possible to suitably transcribe Colonel Maurer's address. Accordingly, the following abstract is presented.

Colonel Maurer presented an illustrated talk on "African Horsesickness" stating, in effect, that after a long history of destruction in Africa, African horsesickness has recently spread to the Middle East. Since 1959, it has killed in excess of 300,000 head of horses, mules, and donkeys in 10 countries in the Middle East. This loss is especially serious there, because of the dependence of the food supply upon equine animals, which provide agricultural power and transportation. As many as 90% of the infected horses and up to 60% of the mules died in some areas. African horsesickness is transmitted by Culicoides midges or gnats which have essentially a world-wide distribution. These Culicoides are efficient vectors, which have demonstrated their ability to transmit this and other diseases internationally. African horsesickness is caused by a virus of which there are many immunologically different strains. Clinically, the disease usually appears in fully susceptible animals as an acute pulmonary edema. A more chronic but highly fatal cardiac form may occur in more resistant animals.

A number of colored slides were shown to illustrate the clinical features and gross lesions of the disease. Colonel Maurer is preparing a comprehensive, illustrated paper on the disease, including the pathology, which will be published elsewhere.

In the 1960 Proceedings of the American Association of Equine Practitioners, Dr. Donald Miller published a paper, which covers the disease quite adequately, apart from Colonel Maurer's illustrations.

XUM

POISONING IN THE HORSE WITH SPECIFIC REFERENCE TO POISONOUS PLANTS

MURRAY E. FOWLER

When a horse dies suddenly and the immediate cause cannot be determined, poisoning, either plant or chemical, is frequently suspected. Poisoning is sometimes used as a "crutch" when no other diagnosis can be made. Many cases of poisoning cannot be determined accurately without laboratory analysis and even then, it is difficult to say for sure that a poison is the cause. People usually do not wish to go to the expense of laboratory analysis and are willing to accept a diagnosis of poisoning. Nevertheless, we should try to make the most accurate diagnosis possible. If litigation is a remote possibility, every effort should be made to obtain an accurate diagnosis. This can be done only if the veterinarian has available certain facts and makes a careful examination utilizing the history, clinical and necropsy findings along with the toxicological analysis. Feeding trials may be used to clinch a diagnosis in some plant poisons.

HISTORY

Is a poison available to the horse? This may not be evident at first. People are not aware of all the possible sources of poisons. Many times a veterinarian has his attention called to poisons either from clinical signs shown, or while looking around the premises. The horse must have access to the poison.

There has to be a sufficient amount of the poison to cause clinical signs or death. There are many misconceptions of the amounts of material necessary to produce poisoning. This is particularly true with plants. Most cases require large amounts to be consumed over extended periods of time in order to cause problems. There are a few, such as oleander or castor bean, where small amounts of the material can cause death. However, in general one can consider that a horse must consume the plant to an extent of 1 to 2 per cent of its body weight in order that toxicity may develop.

Similarly with chemical poisons, more than a gram or two of most chemicals is necessary to produce poisoning. Licking one spot of newly painted material on a board will not cause the death of a horse.

In plant poisonings there must be evidence of the plants having been eaten. It is not enough to find a poison plant on the

XUM

premises. You must be able to show that it has been eaten. Many pastures have plants which are capable of producing poisoning, but do not because the animals will not eat them unless forced to.

CLINICAL SIGNS

Certain clinical signs in a sick horse suggest the possibility of a toxemia. A clinical examination must include a thorough examination of the whole animal, but it will be directed more to certain organ systems. If one were to give a general toxic syndrome, perhaps one would think of an animal with severe diarrhea, with or without colic, associated with convulsions or muscle twitching. The central nervous system, the gastro-intestinal tract and the liver are frequent targets for the action of poisons.

The general signs of the toxemia include anorexia, dehydration, depression and emaciation. The gastro-intestinal signs associated with toxemia include salivation, diarrhea, and/or colic with varying amounts of tenesmus. Heavy metals and certain plant poisons cause severe irritation to the gastro-intestinal tract.

Central nervous system signs include muscle twitching, convulsions, paralysis, hyperexcitability, abnormal movement and coma.

Liver involvement produces two characteristic syndromes; the first being photosensitization. Plants like Agavae cause liver necrosis resulting in the build-up of substances which produce photosensitization.

The second syndrome associated with liver derangement produces central nervous system signs associated with liver cirrhosis. This syndrome is frequently called the "dummy" syndrome because the animal wanders about in a depressed state, bumping into objects, pushing against objects, wandering in circles, etc. Liver function tests may aid in the diagnosis of liver conditions.

POST-MORTEM EXAMINATION

Many cases of poisoning will not be seen until the animal is dead. It is always wise to do a post-mortem examination to see if anything specific can be determined. It should be kept in mind that with poisonings, very few lesions are pathognomonic. Gastro-intestinal irritation, hyperemia and sometimes hemorrhage, are seen frequently.

TOXICOLOGICAL ANALYSIS

With certain chemical poisonings such as lead, arsenic, mercury and copper, it is possible to carry out tissue analysis to determine levels. This is usually an expensive procedure and requires special laboratory facilities. However, if litigation is a possibility, this should be done. It should be determined where help can be obtained if such a situation should arise; all poisons

cannot, however, be detected. There is no specific test for many organic chemicals; the chlorinated hydrocarbon insecticides being an example; all that can be done in such cases is to determine chlorine levels in the tissue (fat).

An approximate idea of the substance to be tested for is valuable for it is not possible to run a battery of tests to pick up all toxic agents. The proper tissues should be provided. This requires some knowledge of the detoxification of the various poisons, but to be safe one should supply urine, some portion of the ingesta, liver and the kidneys — and when dealing with insecticides, some body fat. Two or three hundred grams of the material should be supplied.

Specimens should be handled properly. They should be labelled and each tissue placed in separate containers where they can be identified and then sent directly to the laboratory. This is an absolute necessity in court cases. Plastic freezer bags are very satisfactory for handling these tissues because they are non-reactive and can be sealed quite satisfactorily. Tissues should be kept frozen until they reach the laboratory.

FEEDING TRIALS

If enough of the suspected plant is available it can be collected and fed to "skate" animals. However, the poisonous plants may be toxic only at certain growth stages, or when green and not when dried. Therefore, an attempt to duplicate the environment should be made when running a feeding trial.

Feeding trials are expensive, time-consuming, and usually the practicing veterinarian does not have animals available for these tests. If a complete check is required, however, this would be part of the toxicological examination.

TREATMENT

There are very few specific treatments for poisoning. General treatment should be directed at the following factors.

First, the source of the toxic material should be eliminated. To do this, the feed should be checked carefully for chemical or plant contamination. If the animal is on pasture, it is wise to change the animal to dry feed until the source of the material can be found. There is a possibility of "well-meaning" individuals feeding the animals at odd times or feeding substances that may be harmful, such as oleander clippings. Also, the water supply should be checked for toxic material.

The next step is to remove the material in the intestinal tract. Many poisons will have already been absorbed by the time the veterinarian is called, but if some remain, a laxative may prevent

XUM

further toxic action. Materials that can be used include one to one-and-one-half gallons of mineral oil, and/or a pound of magnesium sulfate. Parasympathomimetic drugs such as Lentin* can be used to stimulate the intestine to expel the material. If the animal is already purging severely, caution is advised because the added laxative effect may be sufficient to kill the animal.

In association with the attempt to remove ingesta from the intestines, inactivation of any remaining poison should be tried. Activated charcoal, Fuller's earth, Bentonite and tannic acid have all been used. They are not specific, but act in a general way to absorb the toxic agents. Following the use of these antidotes, the animal should be given another laxative to make certain that this material containing the toxic factors is also eliminated.

Assuming now that the poison is inactivated and an attempt has been made to remove it, the remaining treatment involves the giving of general detoxicants along with symptomatic treatment. General detoxicants are: Calcium gulconate, 20% (100 to 500 cc.), dextrose, 10 to 50%, (500 to 1,000 cc.) and sodium thiosulfate, 25% (100 to 500 cc.). These agents are considered universal antidotes and not specific for a particular poison. Calcium gluconate and dextrose supply nourishment in addition to their detoxicant effects.

Symptomatic treatment should include sedation in hyperexcitable or convulsive animals. Barbiturates such as pentobarbital may be used. In addition, tranquilizers and chloral hydrate-magnesium sulfate combinations also work satisfactorily.

In cases of severe diarrhea it is necessary to maintain water and electrolyte balance.

A thousand pound horse needs at least 20 liters or 5 gallons of fluid per day to maintain equilibrium. In cases of profuse sweating or diarrhea, the fluid requirements may be doubled. Part of the fluid requirements may be given via stomach tube, the remainder intravenously.

PREVENTION OF PLANT POISONING

Very few plant poisonings respond to treatment. Therefore, if we are to be of much assistance to our clients we should preach prevention. Many times on routine calls there is an opportunity to call the client's attention to some infraction of good management which may save a valuable animal. When working with horse clubs, mention can be made of various aspects of prevention. First of all, the veterinarian should acquaint himself with some of these

*Lentin (Brand of Carbachol Injection U.S.P.) is a product of Merck & Co., Inc., Rahway, N. J.

factors, such as knowing the plants that cause problems in his area. It is a general rule that poisonous plants are not palatable, hence horses will eat them only when forced to by conditions such as insufficient forage or mixing poisonous plants or seeds with hay or grain so that the horse cannot separate the poisonous portion. Putting clippings where the horse normally eats is also a dangerous practice. Exceptionally, horses will acquire a taste for some plants such as bracken or yellow star thistle.

It is always wise to provide the animal with good quality hay. It is rare for a horse to be poisoned from plants in hay, however, there are reports of oleander poisoning where animals have obtained leaves in this manner. It is wise for the horseman to check periodically what he is feeding and to use only the highest quality hay.

The client should be on the alert for trouble spots in his pastures, particularly when feed is in short supply. In many cases, proper pasture management will eliminate the problem of poisonous plants. If grazing is sparse and horses are searching for feed, they may eat such plants. A special check should be made around fences, ditches, springs, lakes, barns and other buildings because these are the places where poisonous plants are most likely to be found.

Lawn clippings should never be fed to horses. Not only will severe fermentative impactions occur, but frequently, leaves from ornamental shrubs, such as oleander are present in lawn clippings and may cause death. Many ornamentals are toxic, even though they are not listed in texts on poisonous plants.

Cuttings should not be placed where horses can get at them. In one case in the author's experience, avocado cuttings were placed in an area where horses were pasturing. Within 24 hours nearly all of the animals in the pasture had violent colic as a result of gastro-intestinal irritation.

Frequently, a veterinarian is called upon to go on trial rides as the official veterinarian, or as a judge. Areas where horses are to be tethered or hobbled and turned loose should be checked for dangerous plants. These horses are frequently very hungry and will eat almost anything. In one case, a group of forest service mules was hobbled and turned loose to graze during a lunch stop; within an hour all the mules were violently ill from eating death camus **(Zigadenus sp.)**.

Small areas of poisonous plants can be eliminated by spraying, digging or mowing. If around a swamp or a ditch, the area can be fenced off.

Horses deficient in salt or other minerals may seek unusual

XUM

feeds to satisfy their appetites. It should be remembered that foals and yearlings are more inquisitive and will more readily eat strange plants. When putting horses on a new pasture, prior feeding will help ensure that, as they investigate their new surroundings, they will be less likely to eat unaccustomed herbage.

OLEANDER POISONING

For many years oleander (**Nerium oleander**), has been a cause of poisoning in animals and man. It is an ornamental shrub which grows best in areas where winters are mild. It is very popular in California. Surprisingly, only a few cases of poisoning occur each year. The leaves are not palatable and horses will eat them only by accident. Most cases occur when cuttings are baled in the hay or are thrown where the horse normally eats his hay. The leaves are poisonous in the green or dried state.

The poisonous principle in oleander is a cardiac glycoside with an action similar to that of digitalis. Forty grams of leaves (about 40 leaves) is a lethal dose for a horse weighing one thousand pounds.

The clinical signs of oleander poisoning are the result of the glycoside's action on the heart. Many types of cardiac irregularities are seen. Partial or complete A.V. blocks are noted in the early stages. These may be rhythmic or irregular. Either bradycardia or tachycardia may occur, but, during the terminal stages, the heart rate is increased to 150 or more. Atrial and ventricular fibrillation are seen terminally.

In fatal cases, the course of the disease usually lasts for 10 hours. Signs are not noted until 4-6 hours after a lethal dose has been acquired.

Other signs which may be seen are, depression, sweating, diarrhea (terminally), cold extremities and dyspnea. The temperature usually remains normal.

At present no specific treatment is available. Symptomatic treatment is indicated. A new drug has been used in digitalis intoxication; this will be tried on experimental cases of oleander poisoning and the results reported later.

CASTOR BEAN POISONING

Castor bean (**Ricinus communis**), is grown commercially in the Southern part of the U.S.A. for castor oil. It is also an ornamental shrub. The bean is the poisonous part of the plant. The poisonous principle is a toxalbumin (phytotoxin). Antibodies can be built up against the toxin. A specific antiserum can be made to treat this condition, however, it is rarely available when required.

Poisoning from castor bean has classically been described as a violent irritation of the gastro-intestinal tract. However, experimental production of castor bean poisoning leads the author to believe that this is not the case. There is a characteristic latent period before signs develop. After the material is ingested, it is usually 8 to 12 hours before the first clinical sign is noted: this is a rapid rise in temperature, peaking at 106 to 107 degrees. Associated with this temperature rise there may be colic, the horse kicking at the belly and pawing with the front feet. He usually goes off feed at this time and as a sequel to the high temperature, may drink copious amounts of water. The temperature will begin to drop — becoming normal in about 24 hours. While the temperature is rising, the pulse begins to increase in rate and depth. The heart beat becomes so pronounced that it is visible ten feet from the animal. The animal begins to perspire rather profusely and there is marked depression. The pounding heart beat, sweating and depression may persist for a number of hours. Terminally, diarrhea may or may not be seen and the horse usually goes down in convulsive seizures. The pulse rate is rapid and there is dyspnea. Death occurs 30-40 hours after administration of the lethal dose. Thirty grams of beans (80-100 beans) is a lethal dose for a horse weighing one thousand pounds.

There is a marked change in the blood picture of a horse poisoned with castor beans. The P.C.V. elevates markedly. There is a severe hemo-concentration and in the W.B.C. differential count there is a definite shift to the left.

The pathological examination of castor bean poisoning reveals a large amount of fluid in the lumen of the bowel, hemorrhages on serosal surfaces and gastro-enteritis.

Usually, only general treatment can be given because antiserum is not readily available.

YELLOW STAR THISTLE POISONING

Yellow star thistle (**Centaurea solstitialis**), grows very abundantly in California and is a noxious weed there and in many other western states. It also is found in more localized spots throughout the United States. Poisoning from this plant could be seen in almost any area of the country.

A more precise name for this poisoning is nigropallidal encephalomalacia of horses, the disease is also called "chewing" disease because of one of the characteristic actions of an animal affected by this poisoning. The condition was first reported in California in 1954.

Yellow star thistle must be consumed for 30 to 60 days before signs appear. Once signs have developed, mortality is extremely

XUM

high. Some cases recover if given satisfactory nursing care, but the prognosis is grave and euthanasia is usually suggested. The onset is sudden. The veterinarian may be called to the ranch to remove a foreign body from the horse's mouth, because these animals act as if they cannot swallow. Some clients will mention that the horse pushes his head into water but seems to be unable to drink.

There is a definite paralysis associated with swallowing and inability to prehend food. Hypertonicity of the facial muscles gives a characteristic "wooden" expression. The muscles around the commissures of the mouth are also hypertonic and the animal frequently opens its mouth as if yawning. The tongue is not completely paralysed and may frequently flop out of and into the mouth. The horse can grasp food and move it around in its mouth but is unable to go through the swallowing sequence. The horse is frequently depressed at first and may act as if there is some incoordination. After a day or two, if the animal is given proper nursing care, these signs will diminish and the animal will regain normal coordination. If the animal is not fed by stomach tube, starvation, of course, will ensue.

Occasionally an animal will develop acute star thistle poisoning associated with convulsive efforts. The horse may walk round in a circle. Differential diagnosis must include foreign bodies in the mouth and pharynx and other encephalitic or paralytic conditions of the central nervous system.

Since vital areas of the brain have been softened and destroyed, treatment is of little value and the animal should be euthanized.

Bibliography

Castor Beans

McCunn, J.: Castor Bean Poisoning in Horses. B. Vet. J. **101** (1945), 136-138.

Clarke, E. G. C.: Poisoning by Castor Seed. B. Vet. J. **103** (1947), 273-278.

Oleander

Wilson, F. W.: Oleander Poisoning of Livestock. Ariz. Agr. Exp. Sta. Bull. (1909), 59.

Yellow Star Thistle

Cordy, D. R.: "Nigropallidal Encephalomalacia in Horses, Associated with Ingestion of Yellow Star Thistle". J. Neuropath. and Exper. Neurol. **13** (1954), 330-342.

Cordy, D. R.: "Nigropallidal Encephalomalacia (Chewing Disease) in Horses on Rations High in Yellow Star Thistle". Proc. A.V.M.A. (1954), 149-154.

GENERAL PRINCIPLES IN RADIOLOGY AND INTERPRETATION – I

WM. D. CARLSON

In order to begin my discussion, I would like to start right off with slides.

Fig. 1-3. This first set of radiographs shows the recommended routine views for all extremities—an antero-posterior and a lateral view. It is imperative to take at least two views. This is because the subject material that we are studying is three-dimensional, and there is no way of knowing if a lesion of either increased density or decreased density is on the outside or inside or within the leg.

We recommend that the radiographs always be positioned on the viewer in a set routine manner. This recommendation comes not only from Colorado State University but also from the teachers of veterinary radiology throughout the country. If, at all times, the radiographs were put up in one routine manner, one would have less difficulty in learning the normal anatomy for each part. The antero-posterior view is put up in such a manner that it would be in the same relative position as when you walk up to a horse to make an examination. The right leg would be to your left, and the lateral aspect of the leg to the extreme left of the viewer. On the other hand, the left leg would be to your right and the lateral aspect to the far right on the view box. We always put the lateral view with the anterior aspect directed to the right regardless of the leg.

To examine the radiograph, it is important that you make a careful study of the entire radiograph. It is necessary to completely examine each and every bone structure, that is, examine all surfaces of every single bone, examine every joint, and if there are two legs, examine one and compare it with the other. You are charging a fairly good fee for this service. Granted, most of it is going into overhead, but you should consider always that the fee is for the professional service. If you just take a glance at the radiograph, you are not fulfilling your professional obligation.

Editor's Note: The designation Fig. 1-3 and similar connotations refer to the illustrations in Dr. Carlson's book, Veterinary Radiology. We acknowledge with gratitude, permission by the publishers — Lea and Febiger of Philadelphia — to reproduce many of these illustrations from Veterinary Radiology.

Where two set of figures are mentioned, e.g. Fig. 15-73 (Fig. 1) the second one refers to the illustration which will be found at the end of the author's address.

XUM

It is essential to have proper exposure factors before a diagnosis can be made. It has been our experience that it is better to slightly over-expose a radiograph than to have it under-exposed for the bony structures. This is because you can always use a spotlight to bring out the structures in the whole bone. You cannot do anything to see the structures on an underexposed radiograph.

Fig. 15-73 (Fig. 1). This radiograph shows a type of condition you ought to be familiar with. There is a radio-lucent area in the cortex of the bone, with a radio-dense object in its center. There is a zone of increased density surrounding the entire lesion which is visible on both sides. This is a typical foreign body reaction in bone. It is a focal area of osteomyelitis which has been walled off by the bone seen as a sclerotic area. This is similar to an abscess in the soft tissues. The material in the center can be nearly the same as the material in the center of an abscess. The object in the center of the radio-lucent area is a sequestrum, a piece of dead bone. We prefer the term "osteomyelitis" if there is an infection of the bone. Modern orthopedic writers in human medicine use the term "osteomyelitis" rather than try to break it down to terms such as "osteitis." I think "osteitis" as generally commonly used in horses is a misnomer. I won't condemn "osteitis" altogether, but I think it should be more carefully used by veterinarians.

Fig. 16-3 (Fig. 2). This slide proves that with the usual radiographic procedures, you can examine the skull of a horse quite satisfactorily. It is nice to have larger equipment for work on the skull, but it is still quite possible to do this with the equipment most of you own. This animal had a pronounced protrusion on the ventral surface of the horizontal ramus of the mandible. The protrusion represented the growth of a tooth root. Due to impingement by an adjacent tooth, there is no room for the proper upward growth of the affected tooth. Because the tooth will continue to grow, it will grow out of the ventral aspect of the jaw.

This might be surmised by clinical examination but by radiography there is no question about what is happening and it is not very difficult to show the owner just what is happening.

Fig. 1-7. As far as I am concerned a spotlight is an essential item in large animal radiographic interpretation. It is not necessary to get this expensive type. All you need is some strong source of light that is encased within a box which confines the light. A hole is made on one side over which you can lay the radiograph for study. The hole should be far enough away from the light source that it will not cause too much heat. You can curl the radiographs and destroy them, if they touch the bulb. I think this is one of the most essential items in the examination of large animal radiographs. A spotlight allows you to see not only small bony protrustions on the surface of the bone (the abnormal new bone prolifera-

tion) but also permits you to see the soft tissues surrounding the bone. Often it is impossible to radiograph a leg and maintain the soft tissue shadows as well as get adequate density of the bone structures.

Again I would like to emphasize the fact that with overexposure, which you are bound to get in some parts and which you should attempt to get rather than underexposure, the spotlight will bring out the structures very clearly.

Another assist in examining radiographs is to cut out all the extraneous light around the radiographs from the unused portions of the viewers. This can be done by use of cardboard stops, film envelopes or other radiographs.

I would like to mention one other thing we use occasionally and should probably use more often. This is a magnifying glass or hand lens. It is very handy for enlarging a certain affected area.

Fig. 2-1. Next I would like to discuss x-ray machines. The machine I am showing is probably a classic, the General Electric D-3 x-ray machine. It is possible to do very excellent work in large animal radiography with this or comparable machines. We use it and are able to take essentially all of our radiographs from the hock joint down and the carpal joint down, at one-tenth of a second.

There are three factors to be considered when dealing with an x-ray machine. The radiographic exposure has to be made at the fastest possible time. In veterinary radiography this is the number one consideration, and it always will be, as far as I am concerned. The second factor, particularly in large animal radiography, is mobility. This machine has a great deal of mobility because it is possible to release the lock bars and the head can always quickly move as the horse moves. The next consideration is the cost. It is possible to buy very expensive radiographic equipment that will make exposures at a very fast time and would also have some mobility, but it is necessary to compromise somewhere in between. I feel that right now, the best compromise is the machine demonstrated here. This machine is rated 85 kvp, 30 ma, and has an electric timer that can give a one tenth of one second exposure. I am not advocating this as the last word in radiographic equipment, but I know that you can do excellent work with this unit. There are new machines coming out. We are just now being approached about new electronic equipment that will be in a small package with a great deal of mobility. The cost of it may be a problem. When they get this type of unit down to where we can afford it, it will be of great help. One of our mobile units is rated at 125 kvp, 300 ma, and will take radiographs at one sixtieth of one second. I know all of you will say, "My, that is a wonderful thing. Why aren't you using it?" We can't because they can't build this kind of equipment with the mobility to follow a horse! It is impossible to get it mobile enough to get the proper radiographs. Many compromises are neces-

XUM

sary, but I do predict that there will be new equipment—probably in the next two or three years—that will be down near acceptable price ranges for use in equine medicine.

This unit (Fig. 2-1), even though rated at 30 ma, is never used by us above 15 ma. The reason is that if we were to use it at the maximum settings of 85 kvp, 30 ma, and one tenth of a second, the machine would not deliver the maximum energy. This is a point veterinarians often do not appreciate. The x-ray companies will not point it out, so that it is necessary to cut back these machines. Use them at maximum kilovoltage, up to 90, but cut back the milliamperage to about one-half of the rated maximum. Then only will you know you are getting the exact exposures that the machine is supposed to be capable of. This will often improve the radiographic picture over what you were getting at maximum settings. Frankly, when all settings are at maximum you just do not know what the machine is doing.

It is quite important that you have a voltage compensator on your machine. This voltage compensator allows you to select a constant input of voltage, no matter where you are, or no matter how many electric stoves are being used down the street. This allows you to select a certain level of voltage that will always be the same. It has been our experience, even at the hospital at Colorado State University where there is an excellent source of electricity, that the voltage will vary enough to change the quality of radiographs on different days. Certainly, if you are using equipment in various places, it is most essential to have some type of voltage compensation so that you have constant voltage going into the machine to obtain some degree of reproducibility at all times.

Portable units often have only one or three kvp settings. I do not know what they represent, but it has been our experience that they usually run around 40, 50 and 60 kvp respectively. Some may run 50, 60 and 70 kvp. You have to realize you are working in the low kilovoltage ranges. On this type of unit the milliamperage can go up to 10 to 15 ma. Again, since this unit should always be used at maximum kvp and if you set it up to maximum ma, we seriously question if you are getting the full milliamperage value. The next thing is a mechanical timer. These timers are inaccurate to begin with and with use, they get worse. If you intend to modify any of your equipment, the first thing you should consider is to get an electrical timer which will give much more accurate timing.

I would like to advocate one other thing—use high kilovoltage technique. This will often not give as pretty a radiograph but it will give a more overall diagnostic radiograph and it will also permit the use of the fastest possible times. In other words, we advocate that you increase the kilovoltage up to 90 and decrease the milliamperage. This also will cause the emission of less total x-rays, which is safer. It is easier on your machine, too. It also

permits a greater margin of error in your calculations. For instance, at the 80 kvp range you could make an error of approximately 8 kilovolts on either side and still have a diagnostic radiograph. In other words, if you were supposed to take the radiograph at 80 kvp and you made a mistake and took it at 72 kvp or 88 kvp, this would still be a good diagnostic radiograph. However, if you were back in the 50 kvp range and you made an error of more than 2 kvp, (less than 48 or more than 52), you would have an underexposed or overexposed radiograph respectively. There are, therefore, great advantages in using high kvp technique. You do not get as high contrast, but you will get a more overall diagnostic radiograph.

Fig. 2-11. This is a drawing of a grid. A grid is a flat, waferlike structure that is placed over the top of the cassette between the animal and the film. It is composed of lead strips placed side by side with little pieces of wood between them. The purpose of a grid is to filter out all of the scattered radiation. As an x-ray beam passes through any material, there is a certain amount of scatter and bounce of the x-rays in all directions. This leads to a great deal of general, gray, fogginess on the radiograph. Scattered radiation is not of great concern until the object to be radiographed is approximately 11 cm thick. This is approximately the size of the average man's knee, which is a rough idea of when a grid should be used. In other words, in large animal radiography its use is necessary in the A.P. view of the hock joint, and on anything above the hock and carpus. It is essential to use a grid on anything larger, or you will be faced with a radiograph that is generally gray, fuzzy and hazy. It is just as if you took a good radiograph and painted over it with a gray film and then tried to make a diagnosis. This shows the 5:1 grid. The lead strips are five times as deep as the space between them. I advocate that you do not buy a grid with a grid ratio any higher than 5 or 6:1. You can get them as far up as 16:1. Every time a grid is used the time must be increased. With a 5:1 grid, you have to double the time, or compensate otherwise. With an 8:1 grid you have to triple or quadruple the time and so on up.

Fig. 2-12 (Fig. 3). This is how an 11 x 14 grid can be used for several sizes of radiographs. A 11″ x 14″ plywood insert, with an 8″ x 10″ hole cut out of it is put in the grid holder. A couple of corner tabs holds the film in. This allows us to use the same grid not only for films measuring 11 x 14 inches but also 8 x 10 inches, which is the size we routinely use on horses. This is just a way of conserving the cost.

Next I would like to discuss briefly x-ray film and screens. Again, I would like to emphasize the fact that we are constantly striving for the fastest possible time. An x-ray screen is another means of improving or speeding up the radiographic exposure. Emulsion is on both sides of the celluloid in the x-ray film. On

XUM

either side of a screened cassette the screens are adjacent to the film. When the x-ray beam strikes the screen, it causes it to fluoresce (to give off a blue light). This blue light exposes the emulsion on the x-ray film. This allows a significant reduction in exposure time. There is a slight reduction in retail, but my personal feeling is that this loss is incidental.

I personally do not feel that there is a lesion so small that you could make a diagnosis on a non-screened film and not be able to make it on screened film. Screened film allows you to take a radiograph much faster. In making a long exposure, which would be necessary with non-screened film, three things can move: the horse, the cassette or rather the man holding the cassette, and the x-ray machine. Certainly, there is much more chance of movement in a one-half or one second exposure than there is in a tenth of one second exposure or less. This movement is usually imperceptible; that is, one cannot tell it happened except that the radiograph looks just a little bit out of focus.

Another factor to reduce the time of exposure is the use of fast or high-speed screens which again does not reduce the diagnostic value of the film. High-speed screens will allow the exposure to be reduced by one-half.

Another way to reduce the exposure time is to use very fast x-ray film. We use Kodak "Royal Blue." Other companies have comparable film, all of which allows the exposure time to be cut essentially in half. "Royal Blue" film requires more care as far as processing is concerned. For instance, it is easier to fog the film, due to leakage of light or due to too bright a darkroom light, but one can easily correct these incidental factors.

These are very important things; I cannot stress them too much. Use high-speed screens and fast film. You then can take the radiograph at probably the fastest setting on your machine.

Fig. 2-16. This brings us into a discussion of labeling of radiographs. The illustration shows the type of labeling that we advocate. You must have your hospital or yourself identified for medico-legal purposes. The next thing is to identify the animal in some way. Each animal should be given a certain number. The date must be recorded. It is necessary and imperative to identify the leg and the surface of the leg. This marker, which probably costs a few cents from any x-ray company, is a plastic plate with lead letters and it says "Lateral Aspect." If you use this lateral aspect marker and if you put it on the film at the lateral aspect of the leg there is no question as to how the radiograph was taken. You can send the radiograph anywhere in the country, and everyone would know what is going on. This refers to the A.P. or P.A. views at which time you need this marker. With regard to the lateral view, it is not necessary to use this marker. The marker "lateral aspect" is also necessary in oblique views.

These markers are taped on the cassette with adhesive tape. All of the markers may be on one piece of tape.

A word about radiographic processing. A full 5-minute development time should be used. This allows the fastest exposure time as well as the best contrast. You cannot satisfactorily compensate an overexposed radiograph by fast development time.

Fig. 15-86 (Fig. 4). This slide demonstrates a most interesting condition which is not seen too often, but you should be aware of it. This was a young colt, with bilateral hind leg lameness. Note that there has been a crushing of the distal row of tarsal bones of both legs. This is a condition that occurs in the human and can occur in all animals. It is avascular necrosis or, in some orthopedic references it is called epiphyseal ischemia, osteochondritis or osteochondrosis. You cannot call it a traumatic lesion. In this condition, the vascularity to some ossification center (this can occur in almost any epiphysis) is lost. The bone then dies. Then circulation from the surrounding structures is re-established. The capillaries invade the area by what is called "creeping substitution." As they invade the dead bone they will reabsorb the dead bone and lay down new osseous tissue which will later become mature bone. However, where the capillaries have invaded a good part of a bone center, the ossification center is not strong enough to support the weight of the animal. At that point, due to the normal weight of the animal, the bone is crushed. Unfortunately, the bone will always remain in the deformed condition. There is nothing you can do. You may be more familiar with it in certain breeds of dogs, or children, where it is called Legg Perthe's disease. (avascular necrosis of the femoral head).

Fig. 15-85 (Fig. 5). The next case is a condition related to the previous case. In this stifle joint of a horse there is a rounded, radio-lucent area on the joint surface of the medial condyle. This is a focal site of vascularity and bone necrosis. The bone died and was re-absorbed by creeping substitution of the capillaries. In the human this condition is called osteochondritis dissecans. We have seen several of these cases in horses. This horse was intermittently quite lame. According to orthopedic texts, indentation of the articular cartilage results. Sometimes in humans, a little piece of dead bone may fall into the joint and form a free joint body. We have not been able to pick these up in horses. When this happens the joint can occasionally become locked and in the human presents severe problems until the piece can be surgically removed.

QUESTION: When does it show up?

DR. CARLSON: We saw these when they were young.

QUESTION: Almost all of these?

DR. CARLSON: Yes, which is also true in the human.

XUM

Carlson; Fig. 15-73 (Fig. 1) Sequestrum in a tibia of a five month old quarter horse filly. Culture of the abscess was sterile.

Carlson; Fig. 16-3 (Fig. 2) Protrusion on the ventral surface of the horizontal ramus of the mandible caused by the downward growth of the root of the second premolar tooth.

XUM

Carlson; Fig. 2-12 (Fig. 3) Showing how the same grid holder can be made to accommodate different sizes of cassettes.

Carlson; Fig. 15-86 (Fig. 4) Aseptic necrosis (osteochondritis). Note the crushed appearance of the distal row of tarsal bones.

XUM

Carlson; Fig. 15-85 (Fig. 5) Osteochondritis dissecans of the medial femoral condyle.

RECENT ADVANCES IN EQUINE ANESTHESIA

E. Wynn Jones

General anesthesia is induced to produce freedom from pain, absence of reflex response to stimulation, and relaxation. The degree to which these effects are required varies with the operative procedure, but in any case maximal safety is essential.

It has been customary in the past to rely upon but one drug to supply these requisites. Examples of anesthetics which have been and still are used for this purpose are chloroform and chloral hydrate.

Chloroform: Advantages of this drug include:

1. It is non-explosive.
2. It is potent and therefore induction and maintenance with minimal equipment is easy.
3. Relaxation is moderate.
4. Respiration of the fetus at parturition is unaffected.
5. Rapid variability of anesthetic depth and recovery is possible.

Disadvantages include:

1. A predisposition to produce cardiovascular disturbances including cardiac arrest.
2. It produces toxic liver damage during prolonged use especially in association with hypoxia and malnutrition.

The disadvantages have therefore caused it to fall into disrepute. It has been mentioned because it is a precursor of halothane and because it should receive further consideration in view of newer developments in anesthetic techniques.

Chloral hydrate: This is probably the most frequently used anesthetic.

Advantages include:

1. It can be administered intravenously.
2. It is relatively safe when used in narcotic doses.
3. Liver damage is not usually a factor.

Disadvantages include:

1. Surgical anesthetic doses cause marked depression of both respiratory and cardiovascular function — a factor in major surgery and operations on poor-risk patients.

XUM

2. Good relaxation requires deep anesthesia.
3. It is a tissue irritant.
4. Recovery is prolonged especially when maintenance doses are used.

To improve the action of chloral hydrate, it was mixed with magnesium sulphate. It was claimed that such a mixture permitted a quicker, more rapid induction with increased anesthetic depth and reduced toxicity. Any real advantage is questionable. More recently, pentobarbital sodium was combined with chloral hydrate and magnesium sulphate. The advantages claimed, include improved induction without excitement, 15-20 minutes of surgical anesthesia following the induction dose, a large margin of safety and quiet recovery without struggling. Prolonged recovery with emergence struggling is still a problem especially following maintenance doses. Relaxation is, however, improved; but respiratory depression characterized by irregular rate and depth is common, even in light surgical anesthesia.

Ether: Although this is considered one of the safest anesthetics for many species, it has always been rather unsatisfactory in the horse due to its inability to produce a sufficiently adequate vapor concentration. Its use is limited although it has been used in Europe with the aid of heated vaporizers.

More recently, tranquilizing drugs have been introduced into equine practice and as a result, the value of pre-anesthetic sedation has become more generally appreciated. The advent of halothane, a chlorinated hydrocarbon, has stimulated interest in inhalation anesthesia, both with this drug and other inhalation anesthetics. Likewise, the widespread use of muscle relaxants has made us appreciate not only the value of these drugs to restrain the horse but also to produce muscle relaxation.

The recent advances relating to equine general anesthesia will be discussed, especially as they relate to inhalation anesthesia and the complications which may arise during the induction, maintenance and recovery.

PRE-ANESTHETIC PREPARATION

Clinical Examination: All too frequently we fail to consider the relation of clinical disease to the induction of anesthesia. Safe anesthesia requires an efficient exchange of oxygen and carbon dioxide between the vital centers and the surrounding atmosphere. This is dependent upon proper respiratory and circulatory function and disorders likely to influence these adversely should be detected and taken into consideration prior to the selection of the anesthetic technique. Such disorders include — limitation of the airway as in roaring, enlarged pharyngeal nodes, nasal discharges,

increased dead space as in broken wind, decreased vital capacity due to abdominal distension, pulmonary collapse or consolidation and abnormal alveolar surfaces for the exchange of gases between the alveolar air and pulmonary circulation. Although cardiac dysfunction poses an obvious hazard, the most frequent circulatory disturbance requiring consideration is shock, whatever its cause may be.

Diet: Food should be with-held 12 hours prior to induction. Bulky foods may be with-held for much longer periods, but extensive fasting is undesirable.

PRE-MEDICATION:

When inhalation anesthetics are to be used, pre-anesthetic sedation reduces nervousness, the amount of anesthetic necessary, facilitates induction, minimizes emergence struggling and assists in the maintenance of an even plane of anesthesia. Depression of the respirations should be avoided especially prior to inhalation anesthesia and in shocked patients. Pre-anesthetic sedatives include — promazine hydrochloride (0.25-0.5 mg per lb) propionylphenothiazine (0.05-0.1 mg per lb) and in cases requiring intestinal surgery, meperidine (0.5-2 mg per lb). Chloral hydrate may also be used (2 gms per 100 lb orally or intravenously).

When irritating inhalants are used, control of excess salivation and mucous secretions with atropine sulphate, (0.02 mg per lb) is advantageous. It is possible that atropine also reduces cardiovascular complications arising from reflex action via the vagus, especially that which occurs during chloroform anesthesia. In view of the developments in inhalation anesthesia and the equipment required for this type of anesthesia, the fundamental requirements for equine inhalation anesthesia will now be discussed.

EQUIPMENT:

The large volumes of air breathed by the horse and the consequent large volumes of anesthetic gas or vapor required, make desirable the use of some type of partially closed system. This is especially important if the anesthetic is expensive (halothane, cyclopropane). In any such system in which expired air is rebreathed, provision must be made for CO_2 absorption. Such systems therefore include:

1. A gas and oxygen supply with pressure regulators and flowmeters.
2. A vaporizer for volatile anesthetics.
3. A CO_2 absorber.
4. A mask and/or an endotracheal tube with adaptor.
5. A reservoir bag.
6. Interconnecting hoses.
7. An exhalation valve.

XUM

1. **Gas and oxygen supply** — For clinic use, piped oxygen or H. cylinders stored some distance from the operation area are preferable. For field use, a small D. or E. cylinder of oxygen is satisfactory. The flowmeter should be graduated from 0-15 liters. For nitrous oxide, similar regulators and flowmeters are suitable. For cyclopropane a small cylinder with a special yoke and a 0-5 liter flowmeter is necessary.

2. **Vaporizers** — A simple bubble-type is very satisfactory for general use, including the administration of halothane. If ether is to be used almost exclusively, an inhaler type in which the animal inhales over the anesthetic is to be preferred. In bubble-type vaporizers, the basal gas flow is utilized for vaporization. Various types of vaporizers, providing controlled vapor concentrations, are available for halothane anesthesia.

3. **Absorber** — This should preferably be cylindrical and 1½-2 times as long as it is wide. It should contain one pound of absorber per 400 cc of tidal air. A 10-12 lb absorber is adequate for the average horse.

4. (a) **Masks** should fit the nose snugly without leaks and should not be larger than is necessary to accommodate the nose. Excessive dead space within the mask is undesirable.

(b) **Endotracheal tubes** should be as large as possible yet still be able to enter the trachea. Neither the tubes nor the tube adaptors should limit the airway any more than necessary.

5. **Reservoir bag** — This accommodates the volume fluctuations as the horse breathes within the closed system. It should be conductive. Too small a bag results in collapse during inhalation and overdistension during exhalation. Too large a bag delays the ability to change anesthetic concentrations and wastes anesthetic. Satisfactory bag sizes range from 20-60 liters.

6. **Connecting hoses** — Should be conductive, flexible, non-kinking, and should exceed the tracheal diameter of animals upon which the equipment is to be used.

7. **Exhalation valve** — This is required to permit exhalation when only partial rebreathing is used, to permit flushing of the system and to prevent excess pressures within the circuit.

Assembly of Equipment

This equipment may be assembled in a circle or 'to and fro' system. In the former, uni-directional valves, to provide a circular flow of air, are necessary.

INDUCTION AND MAINTENANCE:

Fundamentals—To induce and maintain anesthesia, it is necessary to deliver an anesthetic concentration to the brain. This is considered in three phases:

a. Delivery to the animal.
b. Transport to the lungs.
c. Transport to the brain.

A. **Delivery to the animal:** Intravenous anesthetics present no problem. Gaseous anesthetics are delivered by adjusting the flow of oxygen and anesthetic (cyclopropane for instance) to give the necessary concentrations. In the case of volatile anesthetics, it is essential to remember that numerous factors affect the efficiency of vaporization, for example, the vapor concentration varies directly with the gas flow, the temperature of the anesthetic and the surface area of contact between gas and anesthetic which should be as large and as long as practical. Vaporizers should be full, bubbles should be as fine as possible and baffles or wicks are often used to increase the surface area of contact.

B. **Transport to the lungs:** To produce anesthesia, it is necessary to have an anesthetic concentration in the alveoli. Any factor influencing the transport of gases to the alveoli influences the induction, maintenance and recovery from anesthesia. These same factors also influence the gaseous exchange of oxygen and CO_2 and therefore anesthetic safety.

This transport is influenced by:

1. Respiratory minute volume, especially the effective tidal volume.
2. Respiratory obstruction.
3. Variations in vital capacity and dead space.

Assimilation of an anesthetic concentration may be affected by the rate of removal of anesthetic from the alveoli by the circulation. The more soluble anesthetics are removed more rapidly. Concentrations adequate for the induction of anesthesia are therefore slow to develop when using highly soluble vapors such as ether. Induction concentrations are more rapidly achieved with less soluble gases or vapors such as cyclopropane or halothane. Recovery rates are similarly affected.

C. **Transport from the lungs to the brain:** Perfusion of gases across the pulmonary alveolar membranes is adversely affected by any disease condition of this region (edema, fibrosis, pneumonia). Once the anesthetic enters the circulation it passes via the heart to the brain and other tissues. If the peripheral circulation is severely restricted as in shock, a high proportion passes to the brain and induction is rapid. If the peripheral circulation is increased as after eating, a great amount of anesthetic passes to other tissues and induction is slowed. Thus food should be withheld prior to induction; the animal should not be excited and induction should proceed carefully in shocked animals.

XUM

The following fundamentals should therefore be observed:

1. Administer pre-anesthetic sedation and with-hold food to provide quiet, safe induction.

2. Pre-medicate with atropine especially prior to the use of ether.

3. Maintain a free airway.

4. Avoid respiratory depression and increased dead space.

5. Supply adequate oxygen and anesthetic with minimal leakage from the system.

6. When inducing, use a full vaporizer with the anesthetic at room temperature.

Induction

Administer a pre-anesthetic sedative and atropine sulphate if necessary. Intravenous anesthetics are preferred for induction. We usually use 4 mg per lb of thiopental sodium (or equivalent) in 20-25 cc of water, injected rapidly. Administration should be much slower for foals or animals exhibiting shock. Occasionally, chloral hydrate - magnesium sulphate - pentobarbital sodium mixture is used. Alternatively induction can be achieved by:

a. The use of halothane or cyclopropane in a closed circuit once the animal is restrained and recumbent.

b. Restraint with succinylcholine prior to induction with an inhalation anesthetic. Although the hypernea during the recovery from succinylcholine hastens the uptake of anesthetic, we believe there is an unnecessary hazard (at least until more information is available) in inducing anesthesia in the presence of hypercapnia and anoxia, especially in view of heart arrhythmias reported to occur during the use of succinylcholine.

The closed system is checked for leaks, the presence of fresh absorbent, adequate oxygen and anesthetic. The bag is half-filled with oxygen, the exhalation valve closed, and once the animal is restrained or induced, the mask is applied or the endotracheal tube inserted. The gas flow is adjusted to maintain partial inflation of the reservoir bag (average horse — 1-3 liters per minute). If cyclopropane is used, a 50 per cent mixture with oxygen should be used. When using halothane, calibrated vaporizers should be full on; bubble type usually about half-way on. If ether is used, the vaporizer should be full on and the gas flow no more than necessary to prevent collapse of the bag. The necessary concentration of anesthetic in the system can be achieved more rapidly by flushing the system once or twice by opening the exhalation valve and compressing the bag. When induction is complete, the anesthetic concentration should be reduced to maintenance levels. It may be necessary, for short periods, to turn off cyclopropane and halo-

thane. During maintenance, the respiratory and heart rate should be even and regular.

When recovery is desired, the anesthetic is turned off, the exhalation valve is opened, the oxygen flow is increased to 6 or 8 liters or more per minute, and the bag occasionally compressed to "wash out" the circuit. The rate of recovery depends upon the anesthetic used, its tissue solubility and the efficiency of the pulmonary ventilation of the animal.

Of the inhalation anesthetics in use, the most efficient and safe are halothane, ether and cyclopropane, although as previously mentioned, chloroform possibly requires reconsideration.

Halothane

The anesthetic action of this drug was first reported in 1956 and its use in the horse in 1957. Its essential features are:

1. It is volatile, but not inflammable.

2. Its anesthetic potency is probably twice that of chloroform and four times that of ether.

3. Induction and recovery are more rapid than during the use of chloroform or ether.

4. Overdosage is characterized by progressive hypotension and respiratory failure. The heart slows in deep anesthesia and arrhythmias may occur.

5. Although chemically very similar to chloroform, it does not cause liver damage nor does it have the same tendency to produce cardiac irregularities and failure.

6. In view of its expense for large animals, it is practical to use it only in a closed system.

During the administration of halothane to two groups of animals totaling 160 horses, using a closed-type system and inducing by the rapid intravenous injection of a short-acting barbiturate, the following observations were made:

1. Maintenance required from 2.2-8.7 cc halothane per 100 lbs body weight per hour. Amounts were significantly higher for short anesthetic periods under thirty minutes than for periods in excess of one hour. More anesthetic per pound body weight was likewise required for smaller horses. The average amount of halothane used for the average horse for operations one hour or longer was 3.7 cc per 100 lb body weight per hour.

2. The recovery period varied from 15 minutes to 5½ hours. Values in the two groups varied considerably. The recovery period was significantly longer after major surgical procedures than

XUM

after minor procedures. The duration of anesthesia, unlike intravenous anesthesia, did not appear to influence the time required for recovery. Recovery was prolonged by post-operative restriction of limb function (casts or splints, for example).

Complications were:

1. Difficult transition from induction anesthesia to inhalation anesthesia.

2. Difficult maintenance of an even plane of anesthesia in occasional cases, probably due to unsatisfactory pre-anesthetic sedation.

3. Tachycardia and arrhythmias during stimulation of an inadequately anesthetized animal or during depletion of the carbon dioxide absorbent.

4. Occasional over-dosage with bradycardia and marked respiratory depression.

5. Post-anesthetic extensor dysfunction of one or both rear legs in three animals, possibly due to prolonged restraint. This has not been confined to halothane anesthesia.

6. Severe emergence struggling in four cases. Observations appear to indicate that the presence and degree of this problem is directly related to the efficiency of pre-anesthetic sedation.

The chief **advantages** of halothane anesthesia are:

1. Safety due to comparatively few side-effects and ability to vary rapidly the depth of anesthesia.

2. It is non-explosive.

3. It provides fair relaxation in light anesthesia.

4. Recovery is comparatively rapid and usually uncomplicated by violent struggling.

Cyclopropane

This gas was first used in the horse in 1946. It is a potent anesthetic, relatively non-irritant, provides fair muscle relaxation and is a respiratory depressant. The quality of anesthesia is similar to that of halothane. It is highly explosive when mixed with air, oxygen, or nitrous oxide and like halothane is expensive.

Ether

This anesthetic is relatively non-toxic, produces good relaxation without undue respiratory depression. Efficient induction and maintenance in large animals is difficult due to the need for high initial concentrations and a very rapid fall in vapor concentration due to evaporative cooling. In anesthetic concentrations it forms explosive mixtures with air and oxygen. The usual precautions

are therefore essential. Ether can be satisfactory for maintenance, when using a closed system. It is possible that it can be used in a simple, open auto-inhalation system. Because it is highly soluble, induction and recovery are much slower than with either halothane or cyclopropane.

COMPLICATIONS ENCOUNTERED:

These are less likely to arise when anesthesia is induced to the correct depth. We prefer to interpret our anesthesia as too light, too deep, or just right. Satisfactory anesthesia requires the following:

1. No necessity for restraint.
2. Adequate relaxation for the operation to be performed.
3. Regular even respirations — about 5-8 per minute.
4. A regular heart rate and EKG as judged by use of a heart monitor.
5. Surgical stimulation should not cause any appreciable change in the above conditions.

RESPIRATORY COMPLICATIONS:

Faulty respiration, especially if prolonged, causes defective exchange of oxygen and carbon dioxide and their associated effects upon the circulatory system — in particular, increased predisposition to shock and cardiac arrest. Respiratory complications which adversely affect ventilation include:

1. **Obstruction:** This may be partial or complete and is characterized by inadequate tidal exchange relative to the respiratory effort. The hazards of obstruction should be anticipated whenever possible and an endotracheal tube inserted. When this is not possible, it should be corrected by:

(a) The use of an endotracheal tube.
(b) A naso- or oro-pharyngeal tube.
(c) Suction when appropriate.
(d) Tracheotomy where other methods fail.

Relief from the obstruction should be supplemented by administering oxygen and assisting respiration.

2. **Respiratory failure:** This may be of a temporary nature or persistent until corrected. Brief apnea may occur during rapid barbiturate induction, during induction with ether or chloroform, during insertion of an endotracheal tube, as a result of hyperventilation and during the use of succinylcholine.

Persistent apnea may result from overdosage, respiratory obstruction, or may be the first observed evidence of cardiac arrest. Whenever the respirations fail, a heart monitor is both comforting and helpful.

XUM

Treatment should consist of:

(a) The establishment of a free airway, preferably with an endotracheal tube.

(b) The administration of oxygen.

(c) Artificial respiration using positive pressure or positive negative pressure devices.

(d) The use of respiratory analeptics — we prefer pentylenetetrazol intravenously to effect.

3. **Disturbances of rate and rhythm:** These commonly occur during the use of intravenous anesthetics, especially barbiturates. Hypernea may occur during ether induction and in closed circuits in which the CO_2 absorbent is depleted. Slow breathing usually results from deep anesthesia and may be accentuated by the use of barbiturates or meperidine. Respiratory rates as slow as 2-4 per minute for short periods should not cause undue concern. When respirations are slow during barbiturate anesthesia, they are usually irregular so that disturbing periods of apnea occur. Analeptics combat the hypoventilation in such cases but often accentuate the irregular rhythm.

CIRCULATORY ABNORMALITIES:

These include primary and secondary cardiac failure, shock and variations in blood pressure. Three types of cardiac failure are recognized:

1. Complete heart block.
2. Ventricular fibrillation.
3. Asystolic arrest.

In the human, heart block is treated by electrical stimulation or with isoprenoline; ventricular fibrillation is treated by means of a depolarizing current, and massage is used for asystolic arrest. These types of arrest are not uncommon causes of equine anesthetic fatalities. Treatment is ineffective due to:

(a) The failure to recognize arrest immediately.

(b) The lack of available drugs and equipment.

Knowledge of the possible causes and recognition of impending failure will assist prevention. Primary failure may be caused by reflex mechanisms during induction, recovery, or light anesthesia. Surgical stimulation during these phases may initiate such a mechanism. Hypoxia and hypercarbia predispose to such a response. Cardiac failure may also result from over-dosage, inadequate pulmonary ventilation and cardiac pathology. Impending failure is recognized by deterioration of pulse pressure, cyanotic blood and membranes and variations in heart rate and rhythm if

a monitor is being used. The surgeon should be constantly aware of the degree of hemorrhage and the color of the blood and tissues at the operation site. In this way he can learn to judge the status of the blood pressure and gaseous exchange.

Secondary failure usually results from shock, especially during operations for acute intestinal obstructions. In cases of cardiac failure or impending failure:

1. Cease administration of anesthetic; commence artificial respiration.

2. Inject 2-10 cc of 10 per cent calcium chloride in the cavity of the heart, or 1 cc of 1:1000 epinephrine, except in cases of fibrillation.

3. Inject 20 cc of 1 per cent procaine hydrochloride into the auricle in cases of fibrillation, or intravenously to effect in the presence of disturbing arrhythmias.

For adequate safety during equine anesthesia, it is essential to be able to assist breathing and to monitor heart activity.

SHOCK:

This should receive consideration relative to anesthesia because:

(a) It influences the anesthetic dose.

(b) The maintenance of circulation and gaseous exchange are already embarrassed.

(c) It is likely the shock will be accentuated during the course of anesthesia.

The following procedure should therefore be adopted:

(a) Initiate shock therapy prior to induction.

(b) Pre-anesthetic sedatives and anesthetics should be selected to minimize additional circulatory embarrassment.

(c) Pulmonary ventilation should be efficient and assisted if depressed.

Other anesthetic complications which may arise are:

1. Toxic liver damage — chiefly in relation to chloroform anesthesia.
2. Emergence struggling.
3. Nerve injuries.
4. Explosions.

To conclude, at present no one anesthetic is satisfactory for all cases. For short operations use sedation plus the intravenous anesthetic of personal choice or sedation along with an intravenous agent plus ether by auto-inhalation.

XUM

For longer operations use intravenous induction and halothane maintenance.

For poor risk patients use sedation with halothane or ether induction and maintenance.

For the future, it is desirable to achieve better relaxation by developing the equipment to permit very light anesthesia, supplemented by the use of muscle relaxants. Work is required on the circulatory and respiratory effects of succinylcholine. Other inhalant and intravenous anesthetics and electrical anesthesia should be investigated, paying special attention to the rapid variability of effect, rapid recovery, safety and ease of administration.

SELECTED BIBLIOGRAPHY

1. Allen, C. and Reed, W. O.; Inhalation Anesthesia. Proc. Amer. Assoc. Equine Pract. (1950): 171-176.
2. Duncan, W. A. M. and Raventos, J.: The Pharmaco-Kinetics of Halothane (Fluothane) Anesthesia. Brit. J. Anesth., 31, (1959): 302-315.
3. Fisher, E. W. and Jennings, S.: The Use of Fluothane in Horses and Cattle. Vet. Rec., 70, (1958): 567-618.
4. Jones, E. W.: General Anesthesia in the Horse. Ph.D. Thesis, Cornell University, Feb. 1, 1956.
5. Jones, E. W.: Equine Anesthesia — Maintenance by Inhalation Techniques. J.A.V.M.A., 139 (1961): 785-790.
6. Weaver, B. M. Q.: An Apparatus for Inhalation Anesthesia in Large Animals. Vet. Rec., 72, (1960): 1121.
7. Wright, J. G.: Veterinary Anesthesia. 4th ed. Williams and Wilkins Co., Baltimore, Md. (1957): 176-178.

PANEL ON FOAL DISEASES

Chairman: W. O. Reed

Panel: W. R. McGee

J. S. M. Cosgrove

C. E. Hagyard

CHAIRMAN REED: This is the first time that we have had a panel on foal diseases and it should prove extremely interesting if only because the panel members are all top men in their field. After they have made their presentations, questions from the audience will be welcomed.

DR. McGEE: Routine surgical corrections in the foal are not too many in number, so I have chosen two routine procedures which we use — the correction of an umbilical hernia and the repair of a ruptured bladder.

UMBILICAL HERNIA:

Umbilical hernia is relatively common in the foal and perhaps is not considered serious except in sale or show prospects. Hernias are of all sizes and in the majority of cases will disappear with time. However, some do persist and it becomes necessary for the veterinarian to remove them for esthetic reasons or because of the danger from possible strangulation of the contents.

The time for this operation is usually in the fall, after weaning. Exceptions to this are emergency operations for strangulated hernia or the odd foals that will be shipped where veterinary attention is not available.

The technique I would like to illustrate is a very old method ("old fashioned") and I am sure will be considered somewhat primitive by the younger surgeons present. We continue to use it because through continued, constant use by four generations of veterinarians in our organization, it has proved absolutely dependable, safe, simple and fast.

It is efficient regardless of the size of the hernia. There is no risk of infection because there is no invasion of tissue and there are no stitches to slough. It is simple because it involves the manual application of a simple clamp that requires little time and involves no after-care by the veterinarian.

The only possible adverse criticism is that it is neither spectacular nor dramatic.

XUM

I know that many practitioners use the metal clamp for hernia repair. I prefer the wooden clamp illustrated here (Figs. 1, 2 and 3) because it is lighter, it exerts continuous pressure on the hernial sac and periodic tightening is unnecessary. It is also disposable.

The routine procedure of applying the clamp is as follows:

1. Administer 4-5 cc Promazine intravenously.
2. Clip the abdominal area and hernial sac.
3. Cast and tie in dorsal recumbency, then give Relaxans* or Myotal** to a light anesthetic depth.
4. Completely reduce the hernia and set the clamp.
   (a) Set the clamp snugly against the abdominal wall but do not pull the skin too tightly.
   (b) Set the clamp parallel to the belly wall.
5. Secure the clamp with cord or metal clamps (Radiator hose clamps obtainable from a service station).
6. Tetanus antitoxin is given and the foal is turned out in a paddock with another foal for company. In ten days or two weeks, the clamp falls away. Antiseptic dusting powder may be used if any moisture is present during the sloughing stage.

In addition to the correction of routine hernias, I have used this same clamp and procedure with excellent results following the open reduction of strangulated hernias.

RUPTURED BLADDER:

A condition occurring at birth of the foal or shortly after, is rupture of the urinary bladder. Unless a diagnosis is made and prompt surgery instituted, the foal will certainly be lost. On the other hand if the trouble is recognized early and surgery can be performed on a reasonably strong foal, there is every hope for full and uneventful recovery.

The necessary surgery is relatively simple, requires a minimum of equipment and preparation and can be performed under tack-room conditions when necessary.

Percentage-wise, ruptured bladder is not seen in a highly significant number of cases. The important thing is that they can and should be saved when they are encountered.

---

*Relaxans is a product of Pitman Moore Co., Indianapolis, Indiana.
**Myotal, brand of intravenous anesthetic, is a product of Warren-Teed Products Co., Columbus, Ohio.

SYMPTOMS:

Affected foals are usually called to the attention of the veterinarian three or four days after foaling. Symptoms at this time have been or are:

1. Dullness.
2. Inappetence.
3. Periodic straining.
4. Tail-high attitude.
5. Increased respiration.
6. Labored shallow respiration (in the later stages).
7. Convulsions.

DIAGNOSIS:

This is based on the following findings:

1. Normal to slightly increased temperature.
2. Slightly icteric membranes.
3. Icteric index slightly increased but R.B.C., P.C.V. and Hb. level normal or only slightly depressed.
4. Urination has not been observed.
5. Free fluid in abdominal cavity — confirmed by trocharization.

SURGICAL REPAIR:

The abdominal hair is clipped with fine clippers and anesthesia administered. Nembutal serves very well, 10-12 cc being sufficient for the average-sized foal. This must be repeated in extended operations.

The operative area is prepared and the foal placed and held in dorsal recumbency with the rear quarters slightly elevated.

The mid-line incision gives best access to the bladder and is carried well back to the pelvic brim. It is expedient in colts to reflect the prepuce to one side in order to extend the incision far enough back.

When the incision is made, the abdominal cavity should be emptied of as much of the accumulated urine as possible by compression and manipulation of the foal.

The bladder may now be brought to the incision. It is helpful to remove some of the tension of the abdominal wall by slightly flexing the body of the foal thus providing more working space.

XUM

The rupture is more often found on the dorsal surface of the bladder so the latter must be rotated on its long axis to expose the rupture. In length, the tear may be from one quarter of an inch to the entire length of the bladder.

A double row of No. 1 catgut is used to close the bladder.

The peritoneum and connective tissue is closed with a single row of the same material.

The fibrous abdominal wall is closed with No. 2 stainless steel or equivalent. Vetafil is used to close the skin incision.

Post-operative treatment includes blood transfusion (500 cc), electrolyte solution, steroid and antibiotics. Good nursing is essential and the antibiotics are continued for 6-8 days. A catheter in the bladder is not necessary.

Where the diagnosis is made late and the foal appears to be a poor surgical risk for general anesthesia, the operation can be performed using only local anesthesia.

EDITOR'S NOTE:
Dr. McGee then showed a colored motion picture illustrating the technique of this operation.

CHAIRMAN REED: Our next speaker is the first foreign member of our organization. He has a large practice in Ireland and has a very wide experience in the topic which he is going to present. Dr. Maxie Cosgrove from Dublin, Ireland will discuss "Infectious Diseases of the Foal".

DR. COSGROVE:
With your permission I would like to address you on this subject entirely from a practitioner's point of view and not from the usual academic approach of describing the cause, course, symptoms and treatment of each specific or non-specific disease of foals. I also propose for the purpose of brevity to confine myself to diseases most commonly found in the first fourteen days of life.

My reason for this approach is founded on the old and sound argument against "spot diagnosis" despite the fact that our clients frequently expect and judge our ability on making such a diagnosis following our initial or primary examination.

While admitting that certain conditions such as the "sleepy foal" syndrome frequently present classical symptoms which permit early diagnosis of specific disease, to a great extent in the early stages we are capable only of making a broad diagnosis of bacterial or viral infection which may be localized in any of the body systems or generally throughout the body as in cases of septicemia or toxemia. The prognosis at this early stage is best

assessed by the effects on the heart and circulation of whatever infection is present.

Treatment must of necessity be symptomatic until such time as classical or typical clinical signs of specific disease manifest themselves, supported if possible by laboratory findings.

This interval between primary examination and positive identifying findings may on occasion be critical and vital and while the good, painstaking diagnostician may have satisfied himself as to where the infection is located, he is definitely in doubt as to the precise organism to be dealt with; experience however, has shown us some broad principles which help to tide us over this sometimes worrying period and "hold the fort" so to speak.

Generally speaking, high body temperatures associated with respiratory disease are indicative of gram-positive bacterial infection responding to the penicillins, while slightly lower body temperatures associated with gastro-intestinal infection are indicative of gram-negative infection, for which broad-spectrum antibiotics are the choice. The bacteria in urinary infections are also frequently gram-negative and require a similar holding treatment. Most practitioners prefer to play safely and it is usual at initial treatment, whatever the general diagnosis, to combine gram-positive and gram-negative therapy. At this juncture, this address might end as we all know most of our patients respond to this initial treatment and a simple continuance of medication ensures complete recovery. The antibiotics have made us complacement however, and we are going through a very dangerous irreversible period of losing the great art and science of medicine and becoming simply peddlers of proprietary drugs.

Statistics show us that, notwithstanding the wonderful gift we possess of antibiotic therapy, we have not significantly reduced foal mortality either in Europe or the U. S. A. We are constantly being impressed by the claims of manufacturers of new antibiotics pouring on to the market while we forget that those we already employ may be good enough — if we would apply them only after a thorough examination and diagnosis backed up by accurate laboratory findings and the application of, I am afraid a rapidly depleting knowledge of inorganic chemistry and nursing. I put great importance on this latter feature. Hospitalisation with its nursing facilities, frequently state-supported, is one of the many aids we envy our colleagues in the human field, so I ask this question in all sincerity "Is it outside the bounds of possibility that the veterinary profession may one day educate and qualify nurses to assist the profession?" Think how better off we would be if we had a trained observer on the spot, capable of relating the progress of our very sick patients, able to anticipate an emergency, to relieve pain, to ensure correct degrees of heat and

XUM

ventilation and administer medicines on instructions between our visits. Maybe I am dreaming of Utopia, but I am certain there is no one veterinarian who will not agree with me that there are certain farms on which it is a pleasure to work, simply because an effort is made to nurse, however amateurishly, their very sick stock. There are others and the less said about them the better.

Somehow or other we must solve this problem of ensuring good nursing, not by enthusiastic amateurs, but by a trained force, loyal, and part of the veterinary profession. I believe that only then will we make any really significant reduction in foal mortality.

PROPHYLAXIS

We have found, like many of you, that the administration of 1 mega unit of pencillin, ½ gram of streptomycin, and antitetanus serum within hours of birth, gave immense protection to the newborn foal and in our practice we can say with confidence that S. viscosum equi infection, producing the sleepy foal syndrome, no longer occurs in our patients. We can be equally emphatic about the absence of tetanus and acute streptococcal septicemia, but feel this may also be helped by cervical "culturing" of mares 6 days after foaling and administering treatment if necessary for "oral" infections of streptococcal origin. Ethically this form of prophylaxis is wrong for many reasons but on practical grounds it is most effective.

We do not use vaccines on our pregnant mares in order to boost a high antibody content in the colostrum as we are convinced that this form of protection is insufficient to protect the foal born amongst so many others in surroundings which are virtually impossible to thoroughly disinfect.

DIAGNOSIS, PROGNOSIS, LABORATORY FACILITIES

The diagnosis of infection, whether viral or bacterial, of sufficient virulence to develop symptoms of even mild illness, rests basically on the presence of a febrile reaction, supported by corresponding degrees of lassitude or dullness; and also, depending on the system involved, a reduction or absence of appetite, increased respirations, diarrhea, scanty urination and injection of the mucous membranes may be noted. While it is not within the scope of this address to describe methods of examination, a thorough examination demands time and patience and every effort must be made to diagnose the site of the infection, as valuable guidance is obtained from this information and may lead to the identification of any of the well known conditions affecting young foals. Together with a brief description, I will suggest suitable antibiotic therapy for initial medication but qualify these sugges-

tions by emphatically stating that they are subject to confirmation if possible by laboratory sensitivity tests at the earliest possible moment.

With regard to prognosis, examination of the pulse, heart rate and sounds gives the clearest and most definite indication of the virulence of the infection one is dealing with, and equally important, how the individual is standing up to the disease.

Very frequently, however, the pulse and heart rates are grossly accelerated as the direct result of mere restraint necessary in making an examination and one then becomes dependent on the nature of the heart sounds, and pulse volume. These latter two are the best signs on which to base an early prognosis and they continue to be the scoreboard throughout illness. An unembarrassed heart is the most cheerful and optimistic sign in any infection irrespective of the temperature reaction, the mildness of diarrhea, lung or kidney involvement. While the heart may be embarrassed in many ways, in dealing with infections we are concerned primarily with the toxic, degenerative effects of the virus or bacteria on the organ itself, and the secondary effects on the heart of dehydration and electrolytic imbalance.

In treating severe infections, it follows therefore that the rapid identification of the causal organism along with an estimation of the blood picture are both vitally important. To obtain such information necessitates laboratory facilities, without which the practitioner is at a great disadvantage.

While it is not suggested that every febrile reaction in a young foal demands the taking of specimens for laboratory examination, there have been occasions when cases initially appeared simple and straight forward, yet later on in the course of treatment, I wish I had had laboratory assistance. In fact, today one might not be considered too zealous if one took specimens from every sick foal since the odds are that someday the laboratory findings may anticipate and prevent a serious epidemic.

The type of symptoms exhibited indicates the specimens to be taken but as a routine procedure, blood for culture and analysis is worth having. Rectal swabs are invaluable in gastro-intestinal conditions while nasal discharges will often be of assistance in respiratory disease.

Recently I have become very enthusiastic about urine analysis, particularly in diarrheas. The pH values of urine are a valuable indication of the acid-alkali balance which becomes readily upset with electrolyte depletion, the correction of which is vitally important. Very accurate urine pH values can now be ascertained with indicator litmus, a stock of which in my medicine case, I find just as important as a hypodermic syringe.

XUM

SPECIFIC TREATMENT

Apart from antibiotic therapy on which I do not intend to dwell and with which you are all familiar, I would like to discuss for a moment the use of corticosteriods. During the latter part of the 1961 stud season, we commenced using small doses of corticosteroids (5 mg prednisolone) with selected antibiotics and while it is too early to be positive, I believe this substance may prevent the onset of so-called joint-evil. The manifestation of this disease is understood to be peculiar to foals up to six months old, and is evidenced by the sudden marked inflammation of one or more of the articulations and/or tendon sheaths and bursae of the limbs.

I am convinced that the condition is the result of toxemia because in the early stages at least, the synovial fluids are sterile. It is not to my mind an entity of its own, but it may occur during or subsequent to any of the following conditions.

SLEEPY FOAL SYNDROME DUE TO S. VISCOSUM EQUI

The symptoms usually noted are lethargy which increases hourly; slightly raised temperature — about 102.5 to 103 degrees F.; injected, but not markedly so, mucous membranes; slightly increased respirations and pulse rate up to 100 per minute. The foal, in the early stages, continues to suck normally but remains on its feet for only short periods and spends most of the time sleeping. In the latter stages, nephritis becomes apparent and is evidenced by marked tenderness in the loins and the passage of small quantities of urine. The lassitude becomes more marked and the foal usually goes into a deep coma about 48 hours after the onset.

The condition responds to any of the following antibiotics: streptomycin, 1 gram t.i.d., achromycin, 2 grams b.i.d., chloromycetin, 2 grams b.i.d.; as well as to some of the less common proprietary products. In the early stages, any one of these, given by the intramuscular route and continued for at least three days after all symptoms have disappeared, should effect recovery. In the more advanced cases, achromycin or chloromycetin intravenously in 250 mg doses, together with the 2 grams intramuscularly, is favored.

STREPTOCOCCAL SEPTICEMIA

Septicemia due to hemolytic streptococci is frequently encountered. While it is impossible clinically to distinguish with accuracy between the different types of septicemia, it is believed that when the infection becomes established, the most definite symptoms are found in the respiratory tract, and in limb articulations. Both these types are associated with a marked pyrexia, (usually around 105, but occasionally as high as 107 degrees F.).

Treatment here, of course, is penicillin in massive doses. It is usual to commence with five million units of the sodium salt and maintain blood levels with procaine penicillin. The reason for the change to the procaine salt is that the latter is better tolerated locally, and consequently one is more likely to maintain the confidence of the animal and eliminate the fear that frequently arises as the result of irritant, or at least uncomfortable, injections. As already stated, the type of infection is frequently not apparent, and to guard against such shortcomings, the administration of 1 gram doses of streptomycin together with the penicillin should be routine procedure.

ACUTE GASTRO-ENTERITIS

The cause of this condition is invariably attributed to E. coli. As it is highly infectious and very resistant to control unless rgid disinfection is carried out, this creates some doubt that E. coli is solely responsible, and suggests that perhaps salmonella may be considered as a causal agent. This observation is supported by the high incidence of the disease once it has been introduced to farms early in the foaling season, particularly where good hygiene is difficult owing to the use of old buildings where timber structures predominate. In the October, 1961 issue of the Cornell Veterinarian, this view is supported by Bryans, Fallon and Shephard, in an article on Equine Salmonellosis. I noted, in particular, their use of nitrofurazone which is well worth remembering for the future.

While foals at any age may be affected, the condition is most frequently seen in those about two to five days old, and the younger the foal, the more severe the symptoms. The onset is sudden; what may have appeared a few hours earlier to be a perfectly healthy, lively animal becomes dull, listless and dejected, with a profuse, watery, fetid diarrhea; pyrexia in the region of 103.5 degrees F., and increased respiratory and cardiac rates. The patient rapidly deteriorates, depending on the rate of dehydration. The foal usually continues to suck but with decreasing vigor for some four or five hours following the onset of diarrhea, and on this point is based the intensity of treatment. If a foal is sucking reasonably well at the commencement of treatment, one can be satisfied and confident in relying on an intramuscular injection of 2 grams of chloromycetin or a similar amount of achromycin. Response is frequently spectacular, being apparent in as short a period as six hours, at which time a particular point should be made of revisiting the patient. In the last two stud seasons, during which a great number of foals were treated at this early stage, not a single case was recorded in which all serious symptoms had not disappeared in 10 hours. It is strongly recommended that the antibiotic be repeated in 12 to 16 hours, with a final treatment 24

XUM

hours later. The necessity for this was clearly evidenced some four years ago by the death of a foal which had previously responded in less than six hours to initial treatment. The owner was so satisfied with its apparent, complete recovery that he considered further treatment to be absolutely unnecessary. The foal was found dead at morning stables about 48 hours later. Post-mortem examination revealed lesions of acute septicemia with marked ulceration of the stomach and small intestine.

To those cases encountered at a more advanced stage, achromycin or chloromycetin (250 mg) intravenously are administered, together with the usual 2 grams intramuscularly.

I am not really familiar with viral infections but last May, the virus of equine abortion was isolated for the first time in Ireland, so I look forward to some useful information from this convention.

Our biggest worry is the indefinite infections which often defy diagnosis as to the location; but much worse, the causal organism is resistant or insensitive to commonly-used antibiotics. Antibiotic therapy then becomes one of trial and error and one depends on the febrile reaction as an indication of success or failure. At times we are too impatient and expect miracles from these drugs. Too frequently, we find ourselves hopping from one antibiotic to another. Frequently too, we have had cases where we have gone through the whole antibiotic field and in desperation resorted to sulfa drugs — with immediate success. If anything has had a raw deal in human and veterinary medicine, it is this group of drugs. They appear as the poor cousins of the antibiotics but I am certain at times that they are the better workers.

An expensive antibiotic that became available to us about eighteen months ago is a tetracycline for intravenous use, marketed by Hoechst (Germany) under the trade name "Reverin". This we have found extremely useful in resistant gram-negative infections.

Fungal (Monilial) bowel infection occasionally rears its head but usually responds to "Mysteclin"*, 1 to 2 grams t.i.d. Once in a while, as the result of intensive broad-spectrum therapy we find we have upset the bowel flora, thereby permitting staphylococci to predominate at the expense of E. coli with resulting distressing enteritis and diarrhea. In these cases, oral penicillin in doses of 3 to 5 million units per stomach tube every 8 hours, together with fecal bowel culture from a healthy foal brought about recovery.

On the subject of supportive treatment and nursing, it is well worthwhile remembering that, prior to the introduction of sulfa

*Mysteclin (Tetracycline-Nystatin) is a product of E. R. Squibb, Rahway, N. J.

and antibiotic therapy, the older members of our profession were highly successful in the treatment of infections but to get their results they worked harder and longer hours than we are prepared to do today. Their success was built on experience, shrewd observation, diagnostic ability and a vast knowledge of medicine. Little has been recorded of this great fund of information and I regret to say that we appear to be becoming indifferent to its very existence. Quite rightly, great importance was placed on free excretion, particularly of the bowel and kidney; of the use of stimulants in cardiac and respiratory collapse, of bowel sedatives and astringents, of salines and nutrients to make up fluid loss. They dealt in unrefined drugs, many of which had to be compounded in their own pharmacy. Many useful hints and aids in supportive treatment can be obtained in pre-war literature and applied equally well today. To pursue them in this address would be far too lengthy, so I shall refer only to the most important of them.

The administration of fluids whether they be salines, with or without electrolytes, whole blood or plasma is indicated primarily as a result of the reduction of fluid intake and excessive loss in the excretions during illness and secondarily as a result of the hemopoetic or bone marrow depression by some bacteria or their toxins.

The precise requirements of the individual become apparent following blood analysis, and administration is best based on these laboratory findings. These facilities may not be always available, but from experience we can say that every foal not suckling his normal intake needs parenteral fluids. A non-sucking foal requires a minimum of 2,000 cc in 24 hours (incidentally we never force-feed a febrile foal). We usually provide this in the form of glucose-saline given subcutaneously in the pectoral region. In cases of diarrhea, we prefer electrolytic solutions such as Darrow's solution which has a high potassium content. As mentioned earlier, a low urine pH is a fairly accurate indication of potassium depletion. To aid absorption from the site of injection, we add hyaluronidase at the rate of 3,000 international units per liter.

Blood transfusion is indicated when the erythrocyte level is six million or less. Plasma is extremely beneficial in hyperdehydration and is well worth resorting to in extreme cases.

Vitamin depletion occurs — particularly the B group — in gastro-intestinal upsets and should be corrected.

Amino-acids, parenterally or orally, should be administered when marked protein loss is evidenced by obvious loss of condition during the febrile stage and in convalescence.

Corticosteroids are valuable boosts in general collapse provided they are administered in conjunction with antibiotics.

XUM

The stimulants, caffeine and Coramine*, are extremely useful in respiratory and cardiac failure and may well pull the patient through any emergency.

Oxygen administered through a tent or nasal tube sutured to the nostril can be of immense value in respiratory distress.

Heat from infra-red lamps or blower heaters when the environmental temperature is extremely low adds to the comfort of the patient.

Tranquilizers may and often are useful in diarrheas and will even subdue the pain of a twist gut.

CHAIRMAN REED: I feel we are especially fortunate today in having with us on the panel, Dr. Charlie Hagyard of Lexington, Kentucky, who will discuss "Nutrition of the Foal".

DR. HAGYARD:

Unfortunately, very little research has been conducted on nutritional requirements of the equine species. This lack of work is probably because of two factors. About the time that serious research in nutrition was begun, the horse was ceasing to be a valuable part of our national economy. Now the pleasure horses, the Thoroughbred, the Standardbred, the Quarter-horse and the Saddle horse are extremely valuable, and large sums are received by the Federal and various State Governments as the result of taxes put on racing. Research is an expensive venture but, because of monies acquired directly by government from racing, some aid should be given for this investigational work. The other reason for lack of study in this field is, I believe, the fact that we are primarily breeding for racing qualities where rapid growth, size and uniformity of conformation do not mean as much as in species of animals which are raised primarily for food and milk production.

Because of this lack of research, my remarks must be based on personal experience and must unfortunately deal mostly in generalities.

Nutrition for the foal should be considered from the time of conception. The mares should be kept in good condition — neither too fat nor too thin. In most areas, mares that are not suckling obtain enough nourishment, during the summer and fall, from pasture alone. Some individuals require more than this — a problem which can be taken care of quite easily. When winter sets in it is necessary to place the pregnant mare on an adequate grain and hay ration.

---

*Coramine (Nikethamide) is a product of Ciba Pharmaceutical Products, Inc., Summit, N. J.

After the birth of the foal, obviously his chief source of food supply for the first three or four months is his dam's milk. His rate of growth and condition depends on the quantity and quality of milk given by the mare. Quantity is apparently of more importance than quality. It is regrettable that we have no simulated mare's milk to supplement that of poor milkers. Several years ago a leading Harness-horse breeder became interested in producing a simulated milk. Samples were obtained from mares of the lighter classes and from grade draft mares, from mares known to be good sucklers and from those known to be poor one. These samples were sent to one of the leading milk companies for analysis. The results revealed little variation in the various food and mineral elements. The whole matter was dropped, presumably because it would not be commercially advantageous to market such a product.

It is my opinion that, if given a chance, nature does very well in the rearing of young. The foal, like the new-born of most species, is a great imitator and when a few days old will be seen nibbling hay and oats with the dam. His intake of these, as well as grass, increases as he grows and compensatory amounts of grain should be added. I might say here that as long as he has plenty of exercise it is difficult to give a suckling or weanling too much feed.

The best diet for a mare and suckling foal is composed of good quality oats, sound clover hay or a heavy mixed hay of clover or alfalfa and timothy and good pasture. I feel that this type of feed provides all the necessary mineral elements and vitamins necessary for the proper development of the foal. For early foals that arrive before the advent of spring and green grass, there is certainly no objection to giving them A and D Vitamins in the form of Halibut Liver or Cod Liver Oil.

The care of the orphan foal is also a frequently encountered problem. The simplest and best answer is to procure a nurse mare or foster mother. In Kentucky a few men make a business of having them available and charge $300.00 for their use until weaning time. In the event that a nurse mare cannot be obtained, the orphan should be fed either canned or low-fat fresh milk every two hours for the first two weeks after which the interval between feedings may be stretched to every four hours. As soon as the foal shows an inclination to eat solids he may be given small quantities of calf manna and oats. Exercise, too is very important.

I do not believe in the indiscriminate use of mineral supplements. Over-mineralization can produce as many untoward results as under-mineralization. Most foals require neither vitamins nor mineral supplements. It is true that occasionally there are individuals which manifest evidence of faulty growth by rickets, epiphyseal and joint enlargements, etc. I feel that these cases are

XUM

due to faulty metabolism and the inability of the foal to properly assimilate the food he ingests.

For several years a large number of breeders in our area and elsewhere have had frequent soil tests made. Minerals found in deficient quantities are applied to the pastures. Thus, necessary mineral elements are provided through organic forms which is the way nature intended. Unbalanced mineral conditions in the soil can be corrected by following the advice of reputable soil analysts. To me this is the ideal approach.

In summation, from what we now know, I would say — "keep the mare in good thrifty condition, the mare and foal on an adequate high protein diet and provide ample pasture on minerally balanced ground".

CHAIRMAN REED: Now, as you can see by your program we had listed Dr. Horace Davis for "Non-infectious Diseases of the Foal." Dr. Davis is flat on his back with a heart attack. However, he did call me the other night and asked that certain of his topics be discussed. I have asked these gentlemen if they would mind briefing us on those particular topics. I have asked Dr. Cosgrove to discuss barker foals. This is a condition seen very frequently in Ireland and England, and also to some degree in this country.

DR. COSGROVE:

This barker condition is seen in two forms, acute or peracute and mild or subacute. The acute form is called the barker, the subacute is called the wanderer.

The mortality rate in this condition has been as high as 50 per cent. We think we have reduced it perhaps by 10 per cent in the last few years, but it is still a very serious cause of foal mortality. As far as I know, no causal organism or virus has been identified. Mahaffy of the Equine Research Station, Newmarket, suggested that it may be caused by deprivation of blood, due to tying off the cord immediately after foaling, or the premature breaking of the cord after foaling. He contends that when this happens, the foal is deprived of three to five pints of blood which, if the placenta had not been broken or tied off, would have gone into the foal from the placenta.

The symptoms of the barker are: Epileptic fits which may occur within two to six hours of foaling, or occasionally as early as half an hour after foaling. The stud groom is rather pleased that the foal is well nourished, and often has the idea that the foal is really trying to get on his feet. More careful observation will show that these movements are epileptic in type and the foal is incoordinate; he lies on his side, making galloping movements and wild masticating movements of his mouth. He gets into a sweat, is distressed and has tremendously increased respirations.

The attendants usually try and hold down the foal until they get veterinary assistance. Our treatment for this is one of sedation. We formerly used barbiturates. Now we are using an anticonvulsant called Mysoline, produced by the Imperial Chemical Company in England. I am quite sure similar products are on the market here. The drug is available in tablet or fluid form. We prefer the fluid form, one-half ounce of which is given immediately by stomach tube. This drug, even when given orally, is rapidly effective and sedation is evident in ten to fifteen minutes. We wait about half an hour for the drug to take effect. We do not like to put the foals out too deeply, but just quietened down, so that they react to a touch. I think that is where we have been at fault in the past—putting them out too deeply for this can happen with the barbiturates.

We pass a stomach tube, tie it into the nostril and force-feed the foal every two hours with about 2 to 4 ounces of the mare's milk. We observe it constantly for mild sedation; it may come out rather quickly. We have to stay with it, if possible, and keep it sedated. We allow it to come out of the sedation about every five or six hours, watching to see if the convulsive movements return. If they do, we sedate again. Some come out rather nicely, with no more convulsive movements, but there remains always the case that we call a wanderer. This wanderer is an imbecile foal. It is blind; it exhibits no suckling movements of the mouth. You can put your finger into the mouth, and the tongue seems not quite paralyzed but does not seem to do anything about the finger. If you try to feed with a bottle, it is unable to swallow. On these, we have to use a stomach tube and force-feed them as much as we can, every two to three hours at least for the first twenty-four hours. This period of blindness and inability to suck may last for four to six days, otherwise the foal looks all right; he walks around the stable; he is going around by the wall but seldom runs into it. You wonder if he has some vision. He does not do anything convulsive or impulsive. He knows where the mare is; he will wander around and will not lie down. Very often these cases die from fatigue, if you do not persuade them to lie down. You have to put them down nice and gently. Pick them up carefully by placing your arms around the fore and hind legs, and gently lay them on the stable floor and try to prevent any noise or extraneous stimulation that may wake them up when sleeping.

We use both penicillin and broad spectrum antibiotics but we know they are not doing any good. We have tried vitamins, particularly B-12, but again we know it is supportive treatment and not specific. The treatment is entirely symptomatic.

As I said, the cause is unknown. Certainly, Mahaffy's theory is not the answer but it is well worth considering that in the normal mare, one should allow the cord to remain unbroken and

XUM

not be tied off until all pulsation has ceased; thereby you will guarantee three to five extra pints of blood for the foal. We don't know the answer but we do know that we can get at least 50 per cent recovery by symptomatic treatment.

CHAIRMAN REED: Thank you Dr. Cosgrove.

Another topic that Horace Davis suggested for discussion is hemolytic jaundice. This is a common condition and is of serious economic importance. Dr. McGee will discuss this for us.

DR. McGEE:

It is rather short notice to come up with much information but we are all familiar with the basic symptoms of the disease referred to as jaundiced or icteric foal. I think, perhaps, Doll gave it a reasonable name—iso-hemolytic jaundice of the foal—which means that the foal's own serum is agglutinating the cells.

The condition occurs in two forms, an acute or peracute form and a less severe type. The acute case is noted when the foal is eight to twelve hours old. Everything appeared to be proceeding normally after foaling; the foal has nursed very well until perhaps at six hours he is seen to be somewhat weak. He staggers a bit, has some difficulty in getting up to nurse, but the intention and desire is present and he will try to get up and nurse until too weak to do so.

On examination, such foals exhibit all signs of severe anemia. At this time they are not typically jaundiced in any way; that is, there is no particular evidence of jaundice in the mucous membranes or the whites of the eyes. The condition of the membranes is more of a pale or bluish-white condition, inasmuch as the pigment has really not had time to be released from the agglutinated cells. Of course, a foal in this condition has to have immediate treatment.

Often there is not time to cross-match the blood, which should be done if time is available. In emergencies however, I think one is justified in obtaining whole blood if available preferably from a teaser or cold-blooded mare. An attempt should be made to get as far away as possible from the blood lines on the particular farm. This is necessary only in extreme emergency, where the foal is down and cold, the temperature is below normal and it is apparent that death will follow within an hour or so. When possible, cross-matching should certainly be done. Collect samples, cross-match and come as close to the indicated grouping as you can, since the recovery of the foal will reflect the choice. Failure to cross-match may mean an additional load on the kidneys and sluggish removal of the agglutinated cells from the foal's system. Essentially, blood transfusion is the immediate need.

In addition to this, the exchange transfusion of some three or four liters can be accomplished easily, however I think this is relatively unimportant as long as the foal receives blood.

In exchange transfusion, it is necessary to give a little more blood than is taken out. Time is of the essence in the acute case.

The follow-up treatment includes antibiotic therapy to prevent secondary infections, which are almost certain to occur if one fails to follow up with antibiotics.

Many foals go along remaining thrifty and very active until they are five to seven days old when the first indication of this particular condition is the apparent jaundice in the visible mucous membranes which at times become almost a pumpkin yellow. It varies in severity. We find foals where the red blood cell count will drop to 5 or 6 million without exhibiting any physical weakness. Aside from the color, they probably would be considered normal. If they reach this stage where they are eliminating pigment and, as we say, sludge from the blood stream and are not particularly weak and as long as the blood count is normal, transfusion may not be necessary. I believe those foals will do as well without the whole blood transfusion. Transfusion will enable them to eliminate the agglutinated cells quicker if the situation is not aggravated by off-type blood. However, if the red cell count is below 8 or 10 million, I would not hesitate to give blood especially if the foal is not vigorous.

Again, the foal should be given the benefit of antibiotic protection against secondary infection, because it is especially susceptible to enteric and respiratory infections in this condition. Perhaps I should not, but I will say that I have noticed a marked decrease in the occurrence of this particular condition since we quit using the virus made from liver tissue and lung tissue for the prevention of virus abortion in mares. We found it impossible to make that vaccine without including some red cells, and I am sure we were getting some sensitization from the use of that vaccine. All such cases were not of the acute form, in fact, few showed the typical acute iso-hemolytic reaction seen in a mare which is naturally sensitive to another cell type. We did get the same symptomatology to the point where it was occasionally necessary to give transfusions. In those cases, they did not respond as favorably to transfusion treatment as did the textbook case.

In the typical iso-hemolytic foal, due to sensitization to the cell type and parentage proposition, they did respond very cleanly and very definitely to replacement transfusion, or even just ordinary transfusion with the correct blood group. These that we sensitized with the vaccine were more sluggish; there seemed to be, not a definite sensitization, but a sensitization against different cell types. The membranes turned from red to blue to yellow—to two or three different colors before the foal was finally able to eliminate all the so-called sludge from the blood stream.

I am happy to say we were able to get away from a lot of this by the vaccine that we are using now.

XUM

CHAIRMAN REED: The final subject that Horace Davis suggested we discuss is retained meconium in foals. This may sound very simple but it is a big problem and can cause the death of the foal. Dr. Hagyard is going to discuss this briefly for us.

DR. HAGYARD:

Retained meconium is frequently encountered in foals, as most of you know, and sometimes it is quite difficult to correct. The diagnosis is certainly not difficult. The attendant usually gives enemas and if the meconium expelled is not expelled as it should be, he often calls for help. However, a great many of them will give repeated enemas, which only aggravates the condition and causes swelling of the rectal mucosa, making it all the more difficult for the foal to eliminate the meconium.

My favorite treatment, and the one with which I have had most success, is the immediate administration of castor oil—six ounces to start with, and if the desired results are not obtained in seven or eight hours, I give that much more. I have given as high as a quart, believe it or not, over a period of twenty-four hours and the foal came through all right, along with other things!

Here, again, the foal must be treated symptomatically. If in great pain, anodynes and hypnotics should be given. As I said before, repeated enemas should not be used. The introduction of mineral oil every two or three hours will relieve the irritation in the rectal mucosa caused by repeated enemas.

QUESTION: What results are you getting with Diathal?

DR. McGEE: Diathal works very effectively, I think, in foals. I use quite a bit of it but I do not like to keep it up too long. If the condition keeps repeating, I back off after 24 hours. In most cases it does work very effectively in foals, and I think it is very useful. I use about a 5 cc dose, and sometimes repeat it. If the case is severe at all, it will have to be repeated at about four to six hour intervals.

DR. C. H. REID: I would like to ask Dr. Hagyard if he ever used any goat's milk in orphaned colts.

DR. HAGYARD: I have used it on occasion but it has been quite rare because of the fact that goat's milk is not readily obtainable in our section. I think it would be perfectly all right, though.

CHAIRMAN REED: It works all right?

DR. REID: Yes, it works fine. We have access to goat's milk right now.

DR. CARRICABURU: Are those wooden clamps available?

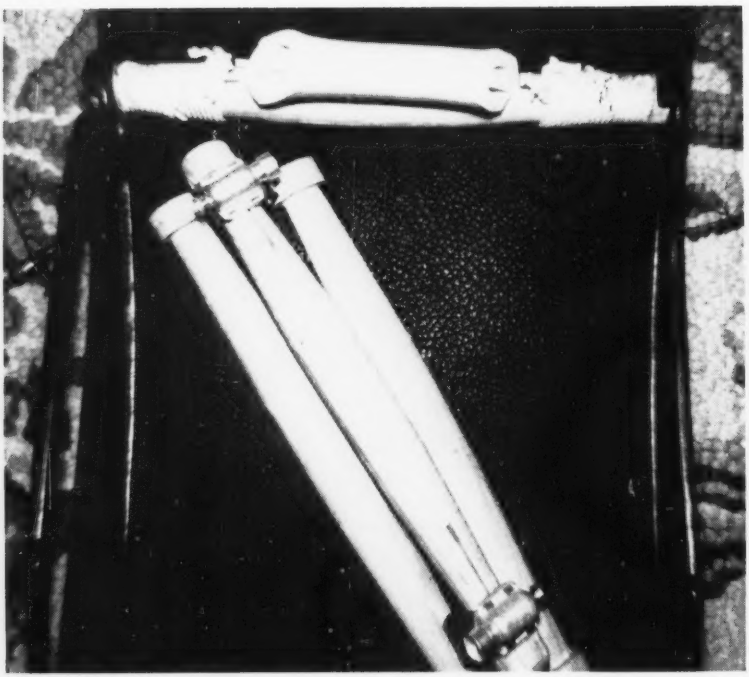
CHAIRMAN REED: How about it, commercially?

DR. McGEE: We have them turned out a couple of hundred at a time.

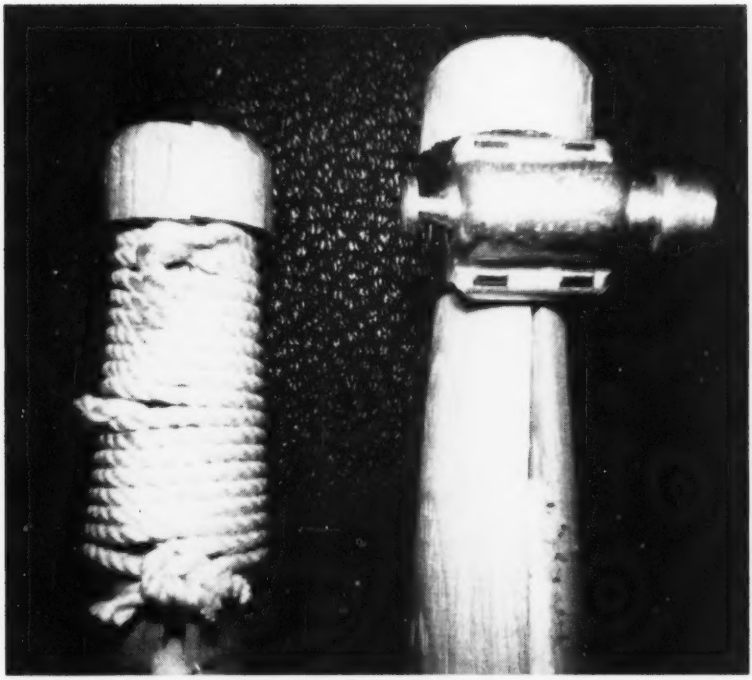
CHAIRMAN REED: Any more questions? It is a real compliment that these topics have been so well covered that there have been so few questions.

McGEE; Fig. 1. Illustrates the wooden hernia clamp, the forceps to hold the clamp in position for tying or placing metal end clamps. Metal hernia clamp facilitates positioning of wooden clamp and is removed when latter is in place.

XUM



McGEE; Fig. 2. Shows forceps holding hernia clamp that has been tied. Another hernia clamp with end clamps secured is shown at center.



McGEE; Fig. 3. Close-up shows detail of two methods of securing hernia clamp.

XUM

GENERAL PRINCIPLES IN RADIOLOGY AND INTERPRETATION – II

Wm. D. Carlson

I would like to begin my remarks with a statement which should stimulate some remarks after my paper. I would like to make a prediction. This prediction is that within the next decade radiation therapy should replace firing as a means of treating lameness in the horse. That ought to stimulate some questions!

I would like to say just a few things about radiation therapy.

There are a few things you ought to be aware of, as far as radiation therapy is concerned. In developing a theme, that it is possible that radiation therapy could become the major means of treating lamenesses, it is going to be necessary that the proper equipment be available in all areas of the country that would be involved in using this type of therapy. In other words, this brings us to a specialized type of practice, a specialty, in which one group of individuals, or one individual, makes the investment and he must be supported by referrals from other colleagues. This is just as true in large animals as in small animals. I sincerely believe that we, as the veterinary profession, could provide a better service to mankind by this type of specialization.

Now, along with the equipment, it is equally necessary that you who are interested in this type of work, receive special training. I am not advocating the extensive residency program as the physicians require. They require four years of postgraduate specialized training to qualify as a specialist in radiology. This, I am not advocating, but I am advocating adequate time spent in training so that you will have some basic knowledge of what you are doing. This will be required of us by the public, which is becoming quite knowledgeable in these matters.

I might be accused of drumming up business for our training program; believe me, I am not. We certainly could not begin to handle it alone.

As far as radiation therapy is concerned, the general opinion is that it will do as much good as firing and, in many cases, much more if it is handled properly. I also feel that the public is going to demand a type of treatment that is not mutilating. I do not believe that any of you can honestly look yourselves in the mirror after you have done a firing job without a little tinge of guilt, because you are definitely mutilating this animal. I am not by any means advocating discarding of firing but I am just presenting the facts. They used to fire people, too! This was discarded

XUM

sometime ago, and if attempted now, would undoubtedly result in a lawsuit.

There has been some misconception as to what radiation therapy will do to new bone growth. Radiation therapy will not remove abnormal new bone growth. I think this is a common misconception among those who are not well acquainted with radiation therapy. What it will do is stimulate or enhance the normal healing processes, which will assist the animal's normal healing of a lesion. Once the stress is removed and the irritation gone, it is possible for these areas of new bone growth to smooth up and round off. This is just normal healing in the animal. This cannot be credited to removal by radiation therapy.

Another very important consideration is that there are different types of therapy machines and different methods of administering therapy. You ought to be well aware of this. If a horse is receiving 1500 roentgens of therapy to 1 cm depth, you must be well aware of the type of machine that is being used, including the half-value layer. When treating a joint of an animal showing a lameness and if using a machine that has an output or potential of 100 kvp, the dose that will reach a depth of one centimeter will be approximately 50 per cent of the dose on the surface. On the other hand, if you are using the unit that we have at Colorado State University, which is 280 kvp, the dose at 1 cm depth would be approximately 85 per cent of the surface dose. If using cobalt 60, the dose at 1 cm would be approximately 99 per cent of the surface dose. You might ask what all this means. It means, when you are treating a lameness condition, you are treating not only the tissues at 1 cm depth but the effects of irradiation are going to be disseminated throughout the entire joint. However, if you are using a low kvp machine or modified diagnostic unit, you could not expect the same type of results from 1000 roentgens as could be expected by using a unit rated at approximately 280 kvp; because, with the latter there will be a tremendous amount of increased radiation in the deeper tissues. In conclusion, when someone tells you he is giving 1000 roentgens to a horse, you had better find out a number of the qualifying factors before you take it at face value.

At Colorado State University, we are using radiation therapy to show our students how this is done and also to try to learn something about what happens. We could treat considerably more animals if we were to encourage the referrals for therapy but we just cannot handle them. We are not trying to drum up business for ourselves. We hope that the practicing veterinarian in the field will take over the bulk of cases.

We are trying to follow up our cases for several years to find out exactly what is happening. We hope to have this information for you, several years from now.

Fig. 15-35 (Fig. 6). This first slide is an example of the type of condition we have been treating with x-ray therapy. This is carpitis with an extensive amount of new bone proliferation on the anterior aspect of the carpal joint. There also appears to be a free body in the joint. This is just an example of one of the cases that we have treated, and it is racing again and winning.

The horses that have had therapy must have adequate rest. We will not treat horses for people unless they guarantee to rest them. I know occasionally they will cheat on us. We feel that rest is an integral part of any radiation therapy we do.

I would like to shift the subject to special radiographic technics. When Dr. Reed spoke to me about presenting this paper, he asked if I could discuss arthrograms and pneumoarthrograms. In order to do this, we had to attempt a number of these at Colorado State University because we were not using them routinely. After you see the examples that I am presenting, I am sure you will appreciate why we are still not using them routinely.

We performed a number of studies using pneumoarthrography, in order to see if we could improve the routine radiographs we were getting.*

The injections were performed under the strictest surgical preparation.

Summarizing the results of our pneumoarthrograms in the coffin, pastern, fetlock, carpus, hock and stifle joints, it is our opinion that these technics were very disappointing. They did not enhance the diagnostic features over the routine radiographs sufficiently to justify the entering of a joint in order to make an injection. Any time the joint space is subjected to an injection, potential trouble exists unless the operator is extremely careful. Pneumoarthrography is not indicated or justified in our opinion.

Fig. 15-7A (Fig. 7). This illustration is presented to demonstrate the problem of trying to locate a chip fracture on the anterior aspect of the carpal joint. It is often necessary to take multiple views at different angles in order to find the fragments. By changing the angulation slightly, we were able to pick up this chip fracture in the case presented. Perhaps a more heavily penetrated radiograph might have shown it through the other structures, probably not.

[Slide] This shows the use of another angle view. This is the angle view to show the lateral angle of the third phalanx. We take a straight view and if there is anything in question in this area of the third phalanx, we take an angle view.

*EDITOR'S NOTE: At this time, the speaker illustrated his remarks with examples of pneumoarthrography technics.

XUM

Fig. 5-38C. This shows the lateral radiograph of the flexed carpal joint. This position allows a more adequate evaluation of the carpal joint. It gives more information than the routine lateral radiograph. If I had my choice, I would take this view in preference to the routine lateral. We take both.

Fig. 15-76 (Fig. 8). This shows another special technic that is of value in selected cases. This is the injection of an infected sinus tract. Occasionally you may encounter a draining tract coming from an area of unknown extent. If you inject some sterile radio-opaque contrast medium under pressure and wash off all the extraneous material, the extent of an infected sinus tract can be clearly outlined radiographically. This is a technic that has limited use, but it is certainly indicated in certain conditions.

Should radiographs be given to the owner? No, it is a dangerous practice to give the owner the x-rays that you take. These are medico-legal records. You are legally bound to have them in your possession. I know you get hounded by people who want their own x-rays; we do too. We have solved this problem by being very accommodating and telling them we will go out and take another set for them at the same price. Some people will go ahead with this, but it quickly puts an end to the requests from most people. Radiographs are legal records, and I do not think you can afford to pass them around indiscriminately. They can be used for illegitimate practices in certain cases.

This brings us into the section of my paper where I want to discuss radiation safety. I would like to set up a situation which I have personally observed and perhaps partaken in, in my earlier days. This shows the operator holding the head of a portable x-ray machine between his legs against his body in order to steady it. The operator, his assistant, the curious bystanders and the owner of the horse are standing around with lead aprons. The encasement of x-ray machines is protected but only for a person at a distance of one meter. It is not intended to provide protection adjacent to the case. We have measured these units with radiation instruments, and there is quite a heavy dose being emitted from the x-ray machine's head. Radiology can be absolutely safe. You really cannot get into trouble **if you take a few precautions.** You have to answer only to yourself for your personal exposure, but you have a man here and you have an onlooker here and you have the owner of the horse there, each of whom you are subjecting to radiation without providing them with the accepted minimal protective procedures which are an apron and gloves. You are letting yourself in for a lawsuit. It does not matter how much scientific evidence there is to say that the amount of radiation that one gets would probably not cause any damage, because this evidence was gathered from mice, rats, dogs and bulls, etc., and didn't come from experimental exposures in man. Without

taking proper precautions, if you were subjected to a suit, I don't think you would have much defence. Some of these suits could be fairly expensive. I do not want to scare you but our courts work that way, because nobody can really say it didn't or couldn't happen to the plaintiff. What do you need for protection? Fig. 5-41A. This illustration shows the proper means of protection. note the aprons, the gloves. People should not be permitted to stand around while you are taking radiographs of horses. I have seen it happen so often in our own hospital, where they will come up and we have to send them out. Watch those little kids. It is your obligation not to let them get around radiography. Fig. 10-2. This next slide shows other examples of gloves and aprons. If you are buying an apron, I advocate the type to the right. This is the type that slips on over the shoulders which makes it easy to get in and out of just like a slip-on apron. The other type has straps which are difficult to get in and out of. Due to these difficulties, it may be easier to leave it off ! Between the two men is a good storage apparatus. Be sure the aprons are stored over a curved surface and the gloves held open. In summary, radiation protection is as vital as any liability insurance you may purchase.

One can wear a radiation film badge. For your own protection, it might be well to provide for a film badge service for a short period of time. If you have a record, a definite record such as this, of the exposures that you and your assistants would get over, say, a three-month period — and it will be far below any of the maximum-permitted levels — it is good proof that you have made definite efforts to monitor and provide safety measures for your people.

Fig. 15-2 (Fig. 9). Some interesting conditions will now be reviewed. The unusual condition that is presented is in a quarter-horse stallion and is called in the human, multiple hereditary exostoses. It is an inherited condition. This animal sired two colts, each of which was affected. Note the protrusions of bone growing at right angles to the cortex of the bone. These protrusions of bone have a cortex themselves that is continuous with the cortex of the rest of the bone. These protrusions also have a medullary canal communicating with the medullary canal of the bone. This is not abnormal new bone proliferation due to injury. This condition is probably due to an arrest of part of the epiphyseal line as it grows out. It can be either bilateral or unilateral.

The condition can affect almost any bone in the body.

This animal also had a protrusion on the rib.

Why do I bring these cases to this meeting? We have seen five cases at Colorado State University. Because this is an inherited condition and was found in good breeding stock, I think you can see the possible problems that could exist. You must be

XUM

aware of this condition and be well aware of the hereditary implications.

Multiple hereditary exostoses causes problems of terminology. The name for this case illustrates the proper use of the term "exostosis". However, we also speak quite indiscriminately of "exostosis" for abnormal new bone growth. In addition we speak often of new bone proliferation as "calcification" which is not true. These radio-densities are generally true ossifications. What I am now showing (Fig. 15-56) (Fig. 10) is rare but true calcification; it is not bone growth. This calcification is confined within a bursa, and is not attached in any manner to the bone. This can also occur in tendons. When you see a radio-opaque density attached to bone, it is not calcification but new bone growth.

Fig. 15-42. Conditions commonly termed "osslets", "ringbone", etc., should be called "abnormal new bone proliferation" or "abnormal periosteal new bone proliferation" according to the teachers of veterinary radiology. Terms such as "calcification" are not recommended.

Next, I would like to discuss several arthritis cases. In the examination of the foot of the horse, you should examine carefully each joint. You will find that the metacarpophalangeal, or fetlock joint is the narrowest joint. The distal interphalangeal joint, or coffin joint, has the widest joint space and the proximal interphalangeal, or pastern joint, is in between the other two joints in width. Awareness of the normal widths of these joint spaces is an important factor, because these regions are where osteoarthritic changes, or hypertrophic degenerative changes develop. The pastern joint is the joint where most often it develops. One of the earliest changes, although they may vary widely in appearance, is a narrowing in the width of the joint. The entire joint usually is slightly narrower, due to wearing away of the hyaline cartilage. If you are aware of the variation in normal joint spaces, this will assist you in determining whether or not there is a decrease in the width of a joint.

Fig. 15-77A (Fig. 11). Notice the narrowed joint space.

In review, in osteoarthritis the joint space generally is narrowed. A narrow joint space and periarticular new bone proliferation are diagnostic signs of hypertrophic degenerative arthritis (osteoarthritis).

These arthritic conditions are believed to be brought on by constant trauma due to poor conformation of the animal. If there is angulation, there will be more pressure on one side resulting in constant trauma resulting in arthritis.

Fig. 15-78 (Fig. 12) This is another case of osteoarthritis. This shows a sub-cartilaginous cystic area. This is not a true

cyst in the sense we think of it being filled with fluid. This cystic area is filled with whirls of connective tissue. This is one of the rarer manifestations of hypertrophic degenerative joint disease (osteoarthritis). Fig. 15-19 (Fig. 13). Here is a case of infectious arthritis. There are very definite radiographic signs that will help you differentiate these two conditions. The clinical picture is an essential factor. We will not discuss it because we are talking about radiography. One of the signs of infectious arthritis is the radiographic appearance of an increased width of the joint. This increased joint space is not due to increased width in the cartilage; it is due to the fact that the infected material has dissolved away all of the cartilage and is now dissolving the adjacent bone (osteomyelitis). This is an infectious arthritis.

To my knowledge, we do not see so-called rheumatoid arthritis in the horse. Rheumatoid arthritis is a collagen disease, and there is no comparable disease in horses.

Fig. 5-46A. Next, let us discuss "navicular disease" syndrome. For proper radiographs the horse's foot is placed on a cassette on the floor, with the toe upon a 2″ x 4″ block. Then the x-ray machine tube is tilted at a 60 degree angle to the floor over the navicular bone. This position will project the navicular bone in a position that is in the central portion of the second phalanx. This view will allow examination of the navicular bone without overlying joint structures. This is the recommended position. The mas should be doubled to show the navicular bone properly. The next position to be demonstrated is an alternative method. (Fig. 5-46B). This employs a horizontal x-ray beam with the foot at a 90 degree angle to the floor and the cassette behind it.

When the navicular bone is to be radiographed, it is imperative that the foot be completely clean and that there be no extraneous dirt on the outside, for the latter will appear as if it were new bone growth. What about "navicular disease" and radiography? Fracture of the navicular bone will cause the same signs as "navicular disease". We see very few fractures, but this is the main reason why we take radiographs in clinical "navicular disease". It is our opinion that one can get positive radiographic signs of "navicular disease" in only about 20 percent of clinical cases. This means that about 80 per cent of the animals showing clinical evidence of "navicular disease" — will not show radiographic signs. Some of the manifestations we would expect to see in "navicular disease" include:

(1) Radio-lucent areas on the distal edge of the bone which are vascular grooves that are increased in size. This has been well studied by our colleagues in Denmark and Sweden and it is their opinion that this is a diagnostic sign of "navicular disease".

XUM

(2) Fig. 15-84A. A cystic area in the center of the navicular bone is another sign. The area of decreased density is a good sign when you see it. However, I suppose we see this particular manifestation in less than 1 per cent of the cases.

(3) Another sign that we will see most often is spurring of the lateral and medial angles of the navicular bone. We see this in various degrees with some even appearing to be rounded. I feel one should question the significance of some of these spurs.

Fig. 15-32 (Fig. 14). The last case I am going to show is a case of rupture of the cruciate ligament. As a result, the spine on the proximal articular surface of the tibia has been broken off. This is believed to be a pathognomonic sign of the ruptured cruciate ligament even though it does not always occur with the condition. This is also seen in man but not so much in other animals.

QUESTION: At what age do you find abnormal new bone proliferation?

DR. CARLSON: Abnormal new bone proliferation occurs at all ages. In review, this terminology describes conditions such as "ringbone", "osselets", "carpitis", etc. The radio-opaque projections that arise from the surface of the bones, as seen in "carpitis", most cases of "osselets", "ringbone" and so on, are all examples of abnormal new bone proliferation. They are not calcifications. We see very little true calcification.

QUESTION: At what age do you consider the epiphysis of long bones to be fused?

DR. CARLSON: I don't know that I can answer that. Bob Adams, can you help out? This is a very variable factor. Just say "NO".

DR. ADAMS: No! (Laughter).

QUESTION: In a two-and-one-half-year-old horse, do you consider non-union of the epiphysis of the tibial crest or distal end of the radius, to be significant as a source of lameness?

DR. CARLSON: No. That has to be qualified a great deal but, in general, no.

QUESTION: In the cases of avascular necrosis that you described in this morning's session, is that condition usually bilateral or unilateral?

DR. CARLSON: It can be either as described in the human. We have not seen enough cases to make any general statements as far as animals are concerned, but I think we can accept pretty well what has been found in the human.

QUESTION: How do you differentiate the cystic areas as seen in arthritis from those of fibrous dysplasia?

DR. CARLSON: Fibrous dysplasia is a developmental lesion in which the cystic areas are of the same type of material; in other words, it is a connective tissue collection within the bone, because of a developmental process; it is a congenital type of process. When you see the cystic areas adjacent to a joint in which there are other manifestations, for example, the joint space is not of proper width, or when you see it adjacent to an osteoarthritic joint, then it must be considered as a manifestation of osteoarthritis. It grows up through the affected cartilage. The damaged cartilage allows this material to grow up into the bone and form these cystic-like areas. I have not seen fibrous dysplasia; it is a rare, rare condition. I have seen lots of such cases in humans. If anybody has one in an animal, I would love to have the case.

QUESTION: What is your technic for stifle examination?

DR. CARLSON: We take a postero-anterior view. We direct the machine from the posterior aspect at an oblique angle of about twenty degrees from the midline out. The contour of the body determines the amount of angulation necessary so that the total stifle joint is on the radiograph. You push the cassette up into the animal's abdomen as much as you can.

QUESTION: What pitch?

DR. CARLSON: It is a straight view; the cassette perpendicular to the floor.

QUESTION: You miss the patella that way?

DR. CARLSON: No. The patella is on the lateral aspect.

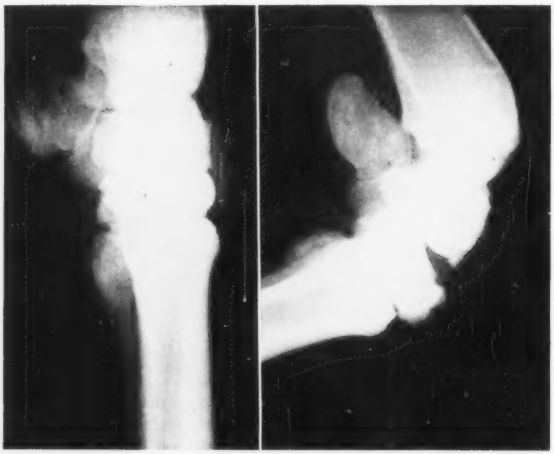
CHAIRMAN REED: Have you done any abdominal x-rays?

DR. CARLSON: Only in very young animals. It is just impossible, with the equipment that is available anywhere in the world, that I know of right now, to do abdominal views. They have a new machine at Ontario that they are working with. I have not seen any of their work. We have done thoracic views. We have a long way to go on any abdominal radiography. We can do the thorax pretty well.

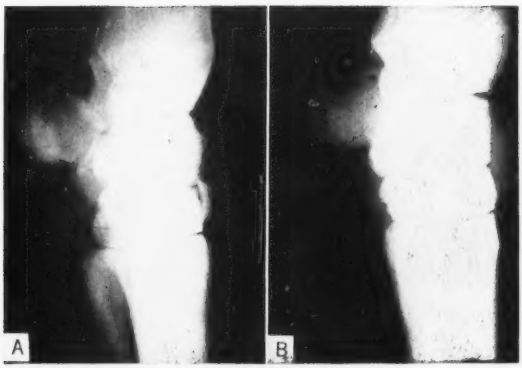
QUESTION: What do you look for radiologically in epiphysitis?

DR. CARLSON: I purposely avoided that. The radiographic signs of epiphysitis, or at least the condition we think is epiphysitis, is an increased radiographic density at the metaphysis. The metaphysis is widened both in the antero-posterior and lateral views. The metaphysis appears to spill over the epiphyseal line, not a great deal, but it appears to hook over like a modified parrot's beak. These are all the possible manifestations. You can see all different degrees of this. This is what we have called "epiphysitis". I don't think anybody really knows what it is. The orthopedic physician will call it epiphysitis.

XUM



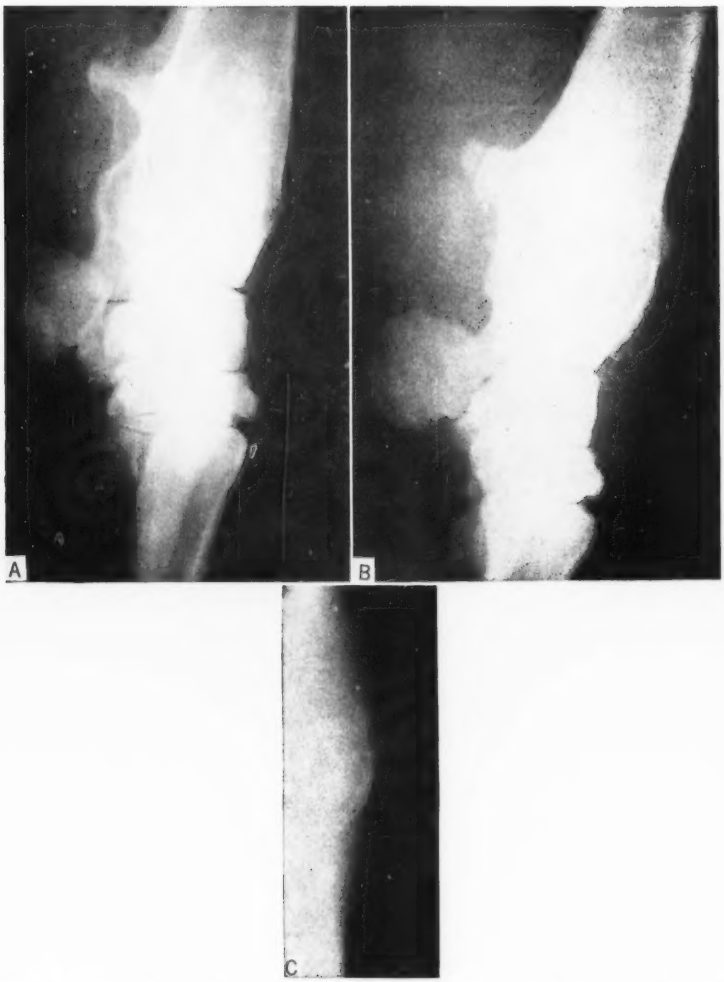
Carlson; Fig. 15-35 (Fig. 6) Carpitis of six months' duration. Note the new bone proliferation on the front of the carpal bones especially on the flexed view.



Carlson; Fig. 15-7 (Fig. 7) Fracture of the third carpal bone. Note that the slightly different angulation of the right hand figure completely obscures the fracture line.

XUM

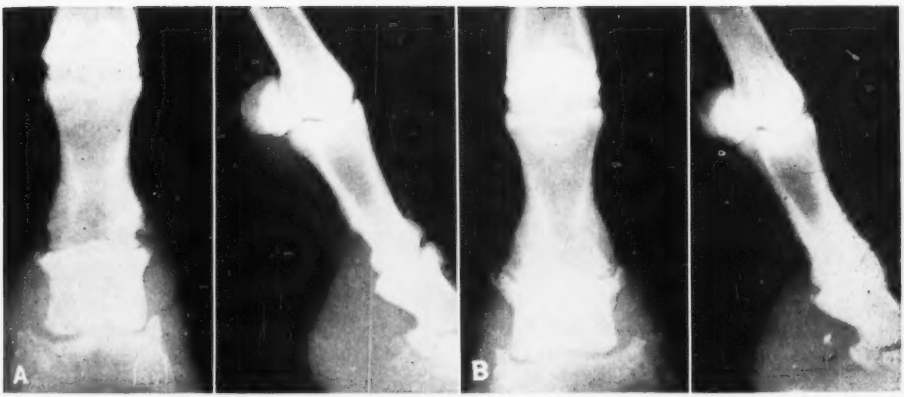
Carlson; Fig. 15-76 (Fig. 8) The injection of a sinus tract. Note the extent of the involved area.



Carlson; Fig. 15-2 (Fig. 9) Hereditary multiple exostoses. (A) Left leg, (B) Right leg and (C) Rib.

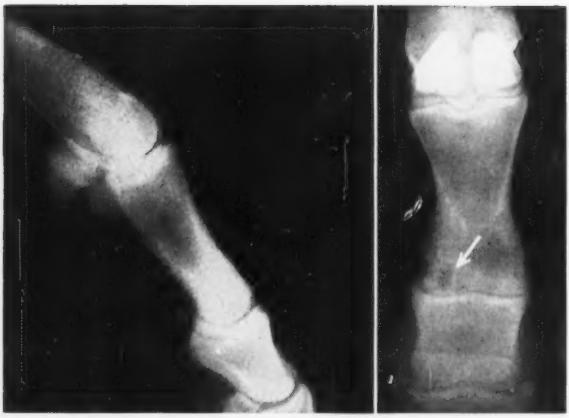
XUM

Carlson; Fig. 15-56 (Fig. (10) Bursal calcification at the fetlock.

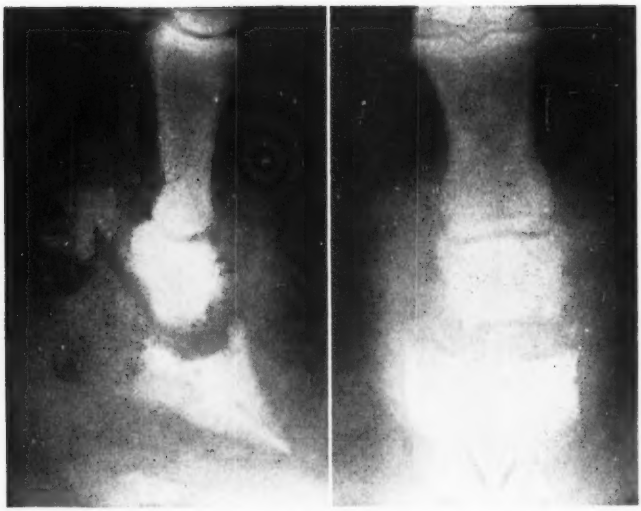


Carlson; Fig. 15-77 (Fig. 11) Bilateral hypertrophic degenerative arthritis (high ringbone). In (A), note the narrowed joint space.

XUM



Carlson; Fig. 15-78 (Fig. 12) Osteoarthritis. Note the subcartilaginous cystic area.



Carlson; Fig. 15-59 (Fig. 13) Infectious arthritis and osteomyelitis. Note the increased width of the joint space.

XUM

Carlson; Fig. 15-32 (Fig. 14) Rupture of the anterior cruciate ligament with fracture of the tibial spine.

EQUINE ABDOMINAL SURGERY TECHNICS

F. J. Milne

At the 77th Annual Meeting of the American Veterinary Medical Association in 1940, the late Dr. James Farquharson[3] while delivering an address on Abdominal Surgery in the Horse said "Little or no progress has been made in surgery of the horse because certain fundamental principles that doom the operation to failure before it started, have been disregarded. It is commonly believed that invasion of the abdominal cavity of the horse is dangerous and unprofitable".

Those words still apply today — the only way in which abdominal surgery of the horse has advanced is the slowly increasing number of practitioners who are willing to take a chance in the hope that success may follow their efforts, yet in the intervening 21 years, man has learned how to harness atomic energy to such an extent that he now has literally at his hand the power to terminate civilization. Such is progress! What a reflection on our present-day civilization!

A study of world veterinary literature shows however, that in an unobtrusive way, equine practitioners are inquisitive about how progress can be made in this field of work. There are many references to successful abdominal surgery in the horse and yet it must be admitted that nobody has yet been able to establish a technic or technics which will cover all the possible situations. This surely suggests that there is a great deal of truth in some of the factors cited as a cause of failure in this type of surgery. Factors such as the size of the patient, its temperament, our relative inability to control the animal during the immediate post-operative period and sutures too weak to stand up to the tension and stresses to which they will be subjected all have an element of truth in them.

Who among us has not had the frustrating experience of performing a surgical intervention on the lower abdomen only to have the sutures break during or after the closure of the body wall with eventration and perhaps evisceration as a sequel? Post-operative straining cannot always be prevented though it may be foreseen, but once it starts we are asking much, and in many cases too much, of our suturing technic.

Perhaps, then, there never will be an answer to some of the problems confronting the equine practitioner. One thing is sure,

XUM

however, we will never succeed if we don't attempt surgery when it is indicated. Only by doing will we learn how to overcome the deficiencies in our knowledge.

My remarks today are taken in part from an article written in the Canadian Veterinary Journal last year by my colleague, Dr. F. D. Horney and myself[7]. This is not a short-course in a capsule form — it is merely a placing before this group of some thoughts gleaned from the literature and from personal experience. I very much regret that the time table of this convention does not permit a full discussion of topics presented, for one learns far more from a discussion than from a lecture, especially when the speaker does not know all the answers.

ANESTHESIA

One of the main deterrents to the performance of this type of surgery in the field is the lack of an efficient, practical and dependable general anesthetic agent for the practitioner working on his own. In the recumbent animal, the practitioner working without professional assistance cannot employ the more modern types of inhalation anesthesia currently being used with great success in some of the European veterinary schools. Reports of agents such as Fluothane are encouraging but are naturally limited to clinics and hospitals, at least at present. In the standing animal the use of local or regional anesthesia precludes sensation but does not guarantee against a sudden, ill-timed movement of the patient when subjected to a separate stimulus. For this reason, there is an element of danger both to the operator and animal when surgery is carried out in the standing horse, however, there are some advantages to this method which will be pointed out later.

PRE-OPERATIVE PREPARATION

We are now living in the period of aseptic surgery, the days of antiseptic surgery having passed into retrospect. With this in mind, I would stress adequate preoperative preparation especially with regard to the operative site. In brief, we can truly say that a swab soaked in antiseptic solution and brushed over the intended site of incision is quite inadequate. It may not be possible to wash the area with hot water and mercurial soap for twenty minutes for three days prior to surgery as did Larsen[5] before carrying out partial cystectomy in a mare, but at least we can shave or close clip and wash the area at least once. Aseptic technic does not end until the operation is completed and the surgeon discards his gloves, hence there must be no breaks in the aseptic chain.

Efficient draping can only be attained if the correct type of materials are employed and the horse co-operates by remaining reasonably still throughout surgery. Drapes which are constantly

being tossed around by a struggling or even restless animal soon lead to a breakdown in aseptic technic. Waterproof drapes in my opinion are far superior to those made of line, cotton, etc., and engender a better sense of security because they do not absorb blood and other fluids from the operation site. My preference is for heavy plastic sheeting. The idea behind the use of Vi-drape* is good but unfortunately the results obtained in large animal surgery have not been as good as those in the human field. This is due to the difficulty of obtaining a firm adhesion between the drape and the skin of the animal, however, the manufacturers are interested in overcoming the problem and I am optimistic that they will come up with something well worth while. The only other drawback is the cost. They are quite expensive.

THE OPERATIVE SITE — SPECIAL CONSIDERATIONS

In the past years there has been a prevalent belief that abdominal surgery in the horse must invariably be followed either by peritonitis or wound breakdown followed by a long period of second intention healing. Such misfortunes followed surgery via the flank for such operations as removal of a retained testicle or a diseased ovary, so one could hardly blame the clinician for not attempting emergency surgery when even elective surgery could end disastrously.

The lack of antibiotic preparations in previous years was blamed for the high incidence of wound breakdown and peritonitis but every one of us here has surely encountered cases where suppuration occurred despite the use of a strong and all-covering antibiotic umbrella. The work of Armistead[1] and others has shown that surgery can be carried out within the abdomen without peritonitis occurring although it must be admitted that the horse seems to lack the walling-off characteristic exhibited by the peritoneum of the bovine species. Veterinary surgeons who carry out operations through the inguinal canal or dorsal fornix of the vagina will agree that peritonitis is a very infrequent sequel to such interventions.

What then are the reasons for wound breakdown following surgery through the abdominal wall? Our belief is that two factors are to blame — (1) struggling of the animal against restraint and also the stress placed on the sutured incision when the animal gets to its feet after surgery; (2) the type of tissue through which the incision was made. The first factor is obvious and we have shown that when a standing laparotomy is carried out, first intention healing is to be expected with a minimum of post-surgical edema. The second factor needs some elaboration. The flank muscles of the horse are very meaty or bulky when

*Vi-drape is a product of Acroplast Corporation — Dayton, Ohio.

XUM

compared with those of the cow and on account of the apparent deficiency of fascia, they lack holding power. It is thus readily apparent that the slightest stress placed on a recently sutured incision where the muscle fibers have been transected is liable to cause disruption, hematoma formation, etc. With regard to the lower abdominal wall where the scalpel will sever essentially aponeurotic tissue, post-surgical edema is an almost-constant feature and is alleviated to some extent only by exercise, but the amount of permissible exercise following major abdominal interventions is insufficient to prevent the formation of this edema.

CHOICE OF SITE

In the standing animal only the upper flank approach on either side is feasible. In the recumbent animal, Marcenac[6] favours a lower abdominal incision. He makes an oblique cut in the lower flank, extending from a point just posterior to the last rib, to a point just in front of the fold of the flank, the direction of the incision being parallel to that of the fibers of the external oblique abdominal muscle. His approach is of the grid-iron type, in that the internal oblique muscle is separated in the direction of its fibers. The transverse abdominal muscle and fascia may be split vertically or in the same plane as the skin incision. Marcenac claims that this site and technic are suitable for abdominal surgery in the horse in that it conforms to the accepted rules of modern surgical technic and assures solid union at the site of intervention. However, it should be pointed out that this type of incision is largely impracticable when the upper flank site is chosen, because the proximity of the last rib to the bony pelvis does not permit adequate exposure of the abdomen unless the incision goes through all layers in the same plane. For the removal of a large mass, such as a foal at the time of Cesarean section, such exposure still may be inadequate. Even at the site recommended by Marcenac, the grid-iron incision may prove inadequate for Cesarean section.

Schebitz in Germany[8] describes what he calls a paramedian "change" incision. This appears to be a modified grid-iron incision. The initial cut in the skin is made eight centimeters lateral to the mid-line. The ventral sheath of the rectus abdominis muscle is cut in the same plane. The author then employs the scalpel handle to divide the rectus abdominis muscle and expose its dorsal sheath. Up until now the incision and division of tissues have been in the same plane but the dorsal sheath is split laterally; by inserting the fingers, the sheath can be torn open towards the linea alba and also further laterally. The sub-peritoneal adipose tissue is removed and the peritoneum is opened during inspiration. The wound is made large enough to admit an arm into the abdominal cavity. Schebitz claims that when abdominal pressure or straining

occurs, none of the viscera will escape past the surgeon's arm. Using this method he operated on 17 horses and was able to bring through the incision all organs which had to be dealt with. No complications followed surgery.

By 1960 Schebitz[9] had employed this approach on 71 cases.

Wheat[10] infers that different conditions will require different approaches. His paramedian approach is carried out in a fashion similar to that of Schebitz. Ellet and his colleagues[2] prefer the mid-line incision as the one that gives the best exposure. Schebitz feels that the mid-line incision through the linea alba is not as safe as one would desire as the fibers within the linea alba do not run parallel to the long axis of the body but are continuations of the three abdominal muscles and therefore run at an oblique angle to the long axis of the body. He further established that by the pull of these muscles the tension on the linea alba is transverse; therefore this type of incision could easily break open during tension, We have favoured the mid-line approach despite these disadvantages because of the good exposure which it gives.

For surgery on the digestive tract, adequate exposure is necessary since it is not always possible to precisely locate the lesion prior to surgery. Certain conditions involving the intestine may demand an exposure in one or the other flank. The inability to bring a diseased portion of bowel to the incision in the belly wall is a frustrating experience which can sometimes be avoided by the use of the mid-line. Where the bowel is tympanitic, it may be necessary to bring some of this distended intestine outside the body wall in order to gain access to the diseased structure. To maintain cleanliness and aseptic technic, the now-exteriorised bowel should be placed on waterproof drapes or sheets. A pervious material, such as is commonly used in shrouds, is unsuitable for this purpose, because capillary attraction permits access of foreign material from the operating table or other environment to the visceral peritoneum. Decompression of bowel may be needed not only to permit location of the diseased portion of intestine but also to allow return of the extraneous mass to the abdomen following correction of the abnormality.

The peritoneum of the equine animal differs from that of the bovine animal in that very frequently a large accumulation of fat is deposited within and around the membrane. Wheat[10] feels that in such cases, the peritoneum should be ignored when the time comes to close the incision. He found that no adverse sequelae follow if it is not sutured. If a mid-line incision has been made right through the linea alba, we feel that in this instance there would be little risk in not suturing the peritoneum. If the peritoneum is laden with fat, suturing of the membrane is contra-

XUM

indicated since fat does not tolerate suturing well, although little objection could be made to closing the gap with fine catgut.

To close the wound we employ the modified Mayo technic; this uses an overlapping mattress (so-called "vest-over pants") suture already familiar to most veterinarians. Double strands of No. 4 medium chromic catgut is the best material for this closure; smaller sizes of material are not advisable on adult horses because they are liable to tear the tissues in the manner of a cheese cutter should the animal move while recumbent. For skin closure, a vertical mattress suture is recommended; one big drawback to this suture is that when the time comes for the stitches to be removed, even though the ends have been left long, difficulty may be encountered in locating the knot because of edema of the abdominal wall in the vicinity of the incision. We feel that where the animal's temperament is unco-operative, the older-fashioned but still serviceable quilled suture is of great value. We use bunsen burner or similar tubing as quills. To remove the sutures all that is necessary is to cut the suture material as it passes over the quill on one side, the entire tubing and severed suture material being easily lifted from the area; there is little upset or irritation to the patient.

When a large incision is called for such as in Cesarean section, the site which gives maximum exposure is the mid-line or paramedian, unfortunately it is a fact that the nearer to the mid-line the incision is made, the greater the risk of herniation. In addition to a careful suturing technic, the suture material employed and the absence of post-operative straining are important. Our experiences with non-absorbable materials in the repair of equine and bovine umbilical hernias prompted us to reject these materials on account of their tendency to form seromas and even suppurating tracts to the exterior. This brought us back to the use of catgut. Despite improvements in standardization, this material sometimes proves to be inconsistent in its tensile strength. Only a few months ago, for instance, we performed and made a motion picture on a perfectly straightforward case of Cesarean section in a 14 year old Palomino mare. This horse should have lived, but eventration occurred late the same evening and on examination it was found that although the knots were still intact, every catgut suture except one had broken. Wright[11] in England has been using a paramedian incision for the removal of a cryptorchid testicle in ponies but there is a great difference between a pony weighing up to 500 lbs and a horse weighing upwards of 1400 lbs. Other workers have failed to substantiate the belief that the paramedian incision is any safer than that made on the mid-line. It is this possibility of post-operative disruption which has steered many clinicians away from the lower abdominal approach. If any other approach is to be used, the severence of muscles across some

of the fibers is inevitable and the holding power of muscle tissue is not great. What of the standing approach for Cesarean section? To my knowledge, only Graden[4] in Switzerland has used this method and he mentioned great difficulty in bringing the foal through the relatively small but maximum-possible size of incision. If we can depend on catgut, I still believe that the mid-line is the answer, but can we place faith in a material which has let us down even in a small number of cases?

In the fortunately rare cases where breakdown of the linea alba has occurred within 48 hours following surgery, the tissues are so devitalized and edematous that resuturing becomes an almost unsurmountable problem. In the two such cases including the disastrous eventration already mentioned, closure was finally effected by using umbilical tape and in each case the final unfortunate result was merely postponed.

Once the symptoms of the acute inflammatory reaction of the tissues have subsided however, repair can more easily be carried out with the tissues better able to sustain the weight of the viscera. This is common knowledge to the large animal practitioner who has had occasion to re-attempt correction of a breakdown following repair of an umbilical hernia.

My colleague, Dr. F. D. Horney, had occasion to operate on a 21-year-old Shetland pony mare. Instead of the anticipated "twist" he encountered a double intussusception — not two separate intussusceptions but one within the other. This involved some 8 to 10 feet of small intestine and was reduced manually. Closure of the abdominal wall was by overlapping mattress sutures. Eleven days later symptoms of acute colic were observed and the mare was returned to the operating room. Entrance to the abdomen was effected through the now almost completely healed linea alba and an impaction of the pelvic flexure was relieved by enterotomy. Closure of the wall was once again carried out by overlapping mattress sutures. Recovery was uneventful.

In conclusion, it is hoped that these remarks will have shown that abdominal surgery in the horse is not impracticable and that more veterinarians will attempt (when indicated) this type of surgery before the prognosis changes from guarded to unfavourable.

REFERENCES

1. ARMISTEAD, W. W. Intraperitoneal surgery in the equine. N. Amer. Vet. 37: (1956) 465.
2. ELLET, E. W., JONES, E. W. and JOHNSON, L. Obstruction of the colon in the horse. Vet. Med. 53: (1958) 291.
3. FARQUHARSON, J. Abdominal surgery in the horse. J.A.V.M.A. 97: (1940) 606.

XUM

4. GRADEN, W. Cesarotomy in two mares. Schweiz. Arch. f Tierheilk, Jan. 1958, 51. Abst. in J,A,V,M,A, 133: (1958) 209.

5. LARSEN, L. E. Laparotomy and cystectomy in a Thoroughbred mare. Aust. Vet. J. 30: (1954) 307.

6. MARCENAC, N. Laparotomie chez les equides. Rec. de Med. Vet. 126: (1950) 129.

7. MILNE, F. J. and HORNEY, F. D. Abdominal surgery in the horse. Can. Vet. J. 1: (1960) 524.

8. SCHEBITZ, H. Der paramediane Wechelschnitt fur die Laparotomie beim Pferd. Berliner und Munchener Tierarztliche Wochenschrift, Vol. 65: (1952) 166.

9. SCHEBITZ, H. Personal Communication, (1960).

10. WHEAT, J. D. Abdominal surgery of the horse. Proc. Amer. Assoc. Equine Practitioners (1958) 77.

11. WRIGHT, J. G. Laparo-orchidectomy in the horse with abdominal cryptorchidism. Vet. Rec. 72: (1960) 57.

EDITOR'S NOTE:

At this stage, the speaker showed several slides illustrating various aspects of his address: one of these is reproduced as Fig. 1.

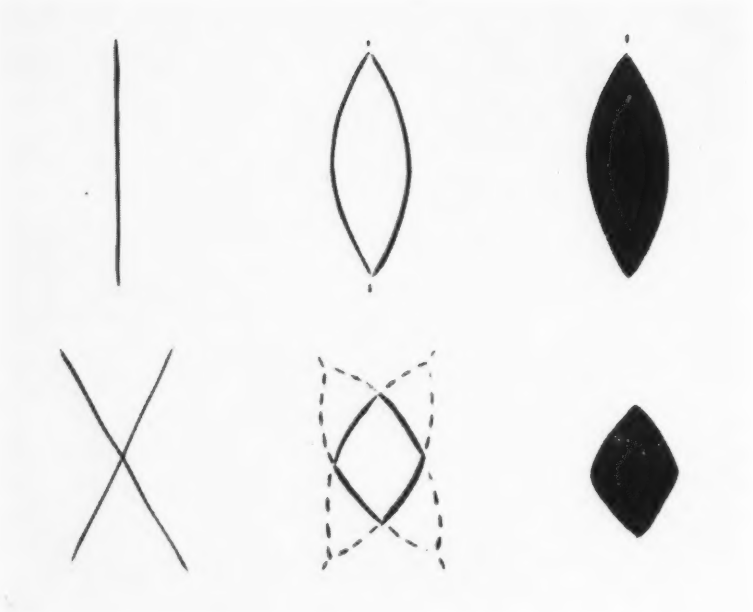
QUESTION: Dr. Milne, I don't know whether one can afford to be as careless with skin suturing in a horse as in a cow. In the cow, using a mid-line incision, we do not worry about the skin sutures as long as we have a good closure of the linea alba. I had a chance to do a comparison a couple of years ago when I was working with people who were placing a tight mattress suture in the skin following Cesarean section while I was placing a very loose stitch which they did not like. The difference in comparison with regard to edema was quite dramatic. Would you comment on that?

DR. MILNE: The question is with reference to post-operative edema and reference is made to the use of a very tight suture through the skin which seemed to increase the amount of post-operative edema. I think it is logical to expect, if you do use too tight a suture, especially a mattress suture, that you are going to kill off tissue just as in the slide which I showed. On the other hand, one is always reluctant to not use the terrific tensile strength of skin. For instance, I rarely use a simple edge-to-edge closure because of gaping of the skin between the loops of suture material; so we try leaving it relatively loose. With a continuous suture we still get edema which products such as the non-mercurial diuretics do not help to diminish. As Dr. Snyder said, if you get edema in a part, you are not going to get as good healing as you would like. I wish someone could help me out on that point.

QUESTION: Does anyone use stainless steel as a buried suture?

DR. MILNE: Stainless steel wire is used by many veterinarians. I believe the California school use wire. The drawback that I see to the use of not only the materials I mentioned, but also to steel is the possibility of operating under unfavorable surroundings. I think that much of the reaction we get in veterinary surgery, especially in large animal practice, to non-absorbable material is on account of the conditions and surroundings we work in. We know nobody can sterilize the human skin. How much farther are we behind the eight-ball in veterinary surgery, especially when sometimes we have to go through encrusted dandruff and dried fecal material before making the incision. Then, too, sometimes the atmosphere in which we are working is polluted when the animal moves or when people come in to watch. I think we run a great risk of later sinus tract formation or infection when we leave non-absorbable sutures in place. From the point of view of breakdown and losing animals, I dislike catgut as I mentioned. Right now I just do not know whether I am coming or going. I know that if I use catgut, I run the risk of herniation and if I use a non-absorbable suture I may get a sinus tract and therefore have to go in and cut out that suture at a later date.

XUM



MILNE; Fig. 1: When operating through the flank, a grid-iron type of muscle incision will give reduced access when compared with that following an incision made in one plane. The illustration shows at top left, an incision made in one plane only. This incision gapes and is changed in form from a line to an elipse (top center). The opening can be further enlarged by means of retractors. At bottom left is shown a "two-plane" incision as in a grid approach. As before, gaping occurs (bottom center) but the size of opening is less than can be obtained following a "one-plane" incision. The illustrations on the right give a rough idea of the degree of exposure obtained following a "one-plane" incision (top right) and the grid approach (bottom right).

EQUINE SYNOVIAL FLUID*

CHARLES E. CORNELIUS

Since articular diseases rank high among the crippling diseases of horses, an understanding of the origin and nature of synovial fluid in health and disease is basic to every equine practitioner. Very little attention has been placed upon the cellular and biochemical changes occuring in synovial fluid of domestic animals. The majority of studies on this fluid has been on the cow[1, 2] with only a few adequate investigations concerned with man[3], dog[4], and horse[5, 6, 7].

The distribution of electrolytes and non-electrolytes between plasma and synovial fluid is in accord with synovial fluid being a **dialysate** of blood, that is, the ions and their distribution are regulated as if a semi-permeable membrane existed between the vascular pool and joint cavity. This is in contrast to cerebrospinal fluid, the aqueous humor of the eye, and urine, which are all actively secreted. Synovial fluid, however, resembles many of the other body fluids in its composition except for the presence of mucin, an acido-glycoprotein. This conjugated protein acts as a lubricant due to its high base combining power and is also quite active in aiding the exchange of water and metabolites between the joint cavity and vascular pool.

It has been suggested that synovial fluid represents a **fluid matrix** of a specialized connective tissue since it is only a dialysate of plasma to which mucin is added as the fluid diffuses through the connective tissue surrounding the joint. The connective tissue which lines the joint cavity has been erroneously referred to as the "synovial membrane". This mesenchymal connective tissue lining the joint cavity is composed of the thin intima and deeper fibrous layer. The underlying articular cartilage lacks regenerative ability in contrast to the unlimited power of repair of this overlying joint connective tissue once infection is removed. The connective tissue lining the cavity has been experimentally replaced in two months after its complete surgical removal.

Theories concerning the lubrication of mammalian joints require a concept of an articular "sponge", not only soaking up fluid and thus ensuring its adhesion to the bearing surface but permitting it to be squeezed out, thus providing an instantaneous formation of a lubricant film with self-movement.

*This is an abstract from a chapter entitled "Synovial Fluid in Domestic Animals" by C. E. Cornelius in **Clinical Biochemistry of Domestic Animals**, Academic Press 1962, In Press.

XUM

EXAMINATION OF SYNOVIAL FLUID

Routine examination of the fluid may include some of the following:

1. **Physical characteristics:** quantity, turbidity, color, specific gravity, clot formation and viscosity.
2. **Cell counts and types:** total W.B.C., R.B.C., and differential counts. If hemorrhage occurs at the time of joint puncture, the ratio of R.B.C. to W.B.C. will be of the order of 500:1. If a relatively greater number of white cells are present, one may conclude that they were present prior to sampling.
3. **Chemical examination:** protein, glucose, acetic acid test, etc.
4. **Bacteriological examination:** Aerobic and anaerobic cultures.

PHYSICAL EXAMINATION

Equine synovial fluid is normally a clear, straw-colored, viscid, tenacious and adhesive fluid which becomes gelatinous upon standing 2-3 hours after its removal. The majority of its inherent osmotic pressure is due to the acido-glycoprotein, mucin, with less than one-third coming from the serum albumin and globulins of the synovial fluid. Mucin has nine times the osmotic pressure as has an equal amount of serum albumin. The pH of synovial fluid is similar to blood and lies between 7.3-7.4. The quantity of fluid varies considerably in each joint.

CYTOLOGICAL EXAMINATION

Fluid should be collected in an anticoagulant as follows: 0.1 ml of 3.8% sodium citrate for 1 ml of joint fluid.

For total cell counts, the undiluted fluid is examined using a white cell counting chamber. If further dilution is needed, one may use physiological saline. Acetic acid should not be used as the mucin will be precipitated. The count is made under high magnification. The total number of cells counted in 9 cm^2 areas on the hemocytometer is multiplied by 10/9 to derive the cells/mm^3. Additional correction for anticoagulant should be made. Rough estimates of the differential count can be made on the hemocytometer if the saline diluting solution contains methyl violet. Average total cell counts in the horse vary greatly from joint to joint per mm^3 — atlanto-occipital, 594(358-1162); elbow, 207(107-336); knee*, 671(390-1638); radiocarpal, 234(40-453); and temporomandibular, 983(412-2350)[5].

Eggers[6] reported from Germany on the results of examination of 20 healthy horses. He centrifuged the fluid and prepared a smear for staining from the last drop in the tube following decantation. Wright's stain can be used to stain the synovial fluid sediment. Differential counts[6] contain: Lymphocytes 10%, young mononuclears 20% and older mononclears 70%.

EDITOR'S NOTE:

*It is presumed that the author is referring to the stifle joint.

An occasional erythrocyte and/or neutrophil may be observed. It is quite interesting to note that up to 10 per cent neutrophils are normally found in the synovial fluid of the cow, man and dog. The presence of a small percentage of neutrophils does not necessarily indicate the presence of infection in a fluid under examination. When erythrocytes are present, they are usually the result of trauma at the time of fluid aspiration, whereas true pathological hemorrhage into joints is accompanied by icterus due to the presence of free bilirubin.

PATHOLOGICAL CYTOLOGY

Information is quite incomplete for the horse. We have observed sterile, clear and viscid synovial fluid with less than 100 mononuclears/mm^3 in traumatic joint effusions. Bacterial infections produce less viscid and cloudy fluids with flocculent particles and at times a count greater than 5,000 pus cells/mm^3. Andreyev[7] has reported that horses with septicemia have synovial fluids with increased mononuclear cell counts. The observation has not been confirmed in experimental neutrophilia in cattle and dogs. Joint ill has long been associated with navel infections in the foal. Descriptions of fluids in 1919 (**Shigella equirulis** infections) stated these fluids were purulent, dark in color, contained air bubbles, were frothy, and contained entangled masses of coagulated fibrin and mucin. Eggers[6] recently reported neutrophil counts up to 1 million/cm^3 in acute infectious synovitis.

CHEMICAL CHARACTERISTICS

Mucin — this lubricant of synovial fluid is an acido-glycoprotein of which the polysaccharide moiety is hyaluronic acid. By adding acetic acid to the fluid, this mucin precipitates into a tough, ropey mass and settles to the bottom of the test tube. It varies in concentration between 100-600 mg per cent. The mucin of the carpal fluid of the horse has a lower degree of polymerization than in other mammals, therefore, it is slightly less viscous than in other species.

Mucin becomes depolymerized or broken down by enzymatic activity in infective bacterial arthritides. This produces a fluid of lower viscosity and the mucin will not precipitate upon the addition of acetic acid, but will become cloudy. Traumatic joint effusions, however, usually maintain their normal concentration, quality of mucin, and hence reaction with acetic acid. Other serum proteins in synovial fluid increase in concentration in joint involvements.

CLASSIFICATION OF PATHOLOGICAL FLUIDS[2]

Group I — **Fluids produced by effusion from traumatic origin.** The fluid is usually clear, does not clot and contains no great increase in leukocytes, which are of the mononuclear type. Mucin precipitated by acetic acid is ropey and the supernatant solution is clear. The

XUM

reduction in viscosity is minimal and the sugar levels are not greatly lowered. Degenerative joint disease (osteoarthritis) and trauma are the main etiological agents.

Group II — **Intermediate Fluids.** Some fluids will exhibit some characteristics of both groups I and III. These may be from cases of arthritis or hydroarthrosis which at first may show a great inflammatory and exudative reaction and later return gradually to normal.

Group III — **Septic Fluids (infectious arthritides).** These show evidence of exudation with marked elevations in the total protein and neutrophils. The mucin may not precipitate or may appear friable upon the addition of acetic acid, and the supernatant solution will be cloudy. The viscosity and sugar levels are greatly reduced.

NON-ELECTROLYTES

Most of these chemical entities are in the same concentration as in blood plasma or at least of a concentration in accord with the theory that this fluid is a simple dialysate of plasma. Non-protein nitrogen constituents are nearly identical in concentration to those of plasma. Glucose is only slightly lower, and this may be due to the glycogenolysis following struggling of the animal during the removal of a sample. Cholesterol and lipids are not present unless pathological changes exist. **Electrolytes** agree in concentration with the existence of a Gibbs-Donnan equilibrium between plasma and synovial fluid.

SUMMARY

Biochemical changes in the composition of synovial fluid produced by pathological conditions involving the joints, depend primarily upon two alterations:

1. Altered permeability of adjacent tissues.
2. Disturbances in intra-articular metabolism.

Experimental studies have shown that bacteria gain entrance to joint fluid more readily than into spinal fluid, aqueous humor or urine. Changes in the permeability of adjacent joint tissues allow for the entrance of water, fibrinogen, leukocytes, antibody globulin and some enzymes. Alterations in joint tissues and synovial lymphatics lead to a diminished removal of particulate and colloidal material from the cavity. The inability to remove these colloids which gain entrance during an inflammatory process increases the osmotic pressure and the quantity of joint fluid.

The use of a few rapid laboratory procedures on the synovial fluid will aid the equine practitioner in arriving at a diagnosis as well as a prognosis in both traumatic and septic arthritides. These should include the total cell count, differential cell count and acetic acid test for mucin.

Table I. Cell Counts from Synovial Fluids of Clinical Cases*

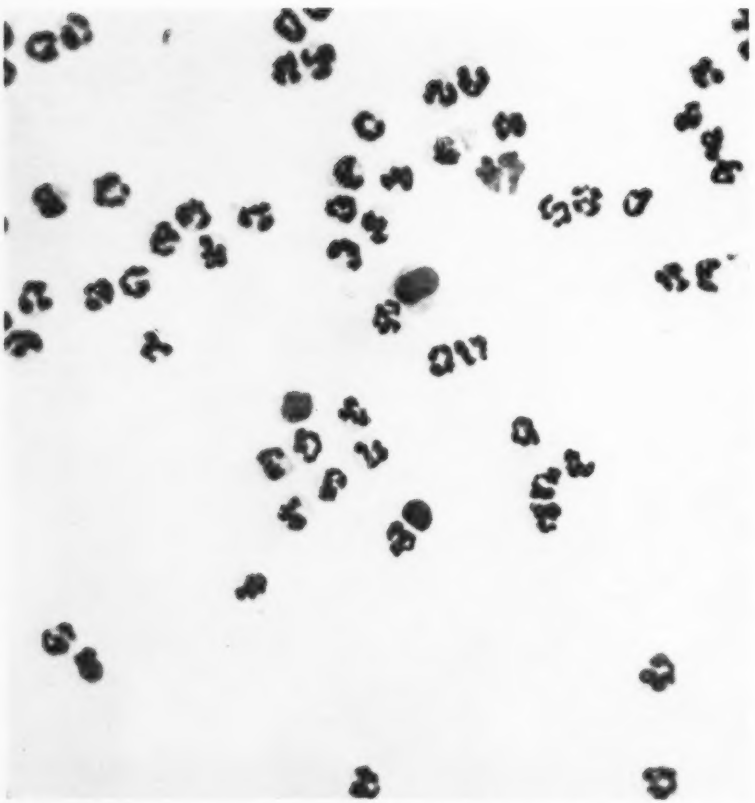
Species	Diagnosis	Fluid	Laboratory Findings
Equine	Osteoarthritis	Carpal	Turbid, yellow fluid; occasional R.B.C.; 110,000 polymorphonuclear leukocytes/mm^3 and no bacteria on culture.
Equine	Traumatic carpitis	Carpal	Clear, light yellow, viscous fluid; 290 leukocytes/mm^3 with a differential count of: polymorphonuclear leukocytes, 4%; small mononuclears, 15%; and large mononuclears, 81.0%.
Equine	Traumatic carpitis	Carpal	R.B.C. 3700/mm^3; and W.B.C. 700/mm^3 (polymorphonuclear leukocytes, 4%; large mononuclears, 16%; and small mononuclears, 80%).
Equine	Arthritis	Stifle	Light yellow fluid with numerous white flakes of fibrin; R.B.C., 50/mm^3; W.B.C. 170/mm^3; with a differential count of small mononuclears, 50% and large mononuclears, 50%. No bacteria on culture.
Bovine	Purulent synovitis	Carpal	Yellowish, opaque fluid with a specific gravity of 1.018; R.B.C., 4100/mm^3; W.B.C., 46,700/mm^3; with a differential count of polymorphonuclear leukocytes, 89%; small mononuclears, 7%; and large mononuclears, 4%.
Equine	Tendosynovitis	Carpal	Light yellow, slightly cloudy, and moderately viscous; R.B.C., 970/-mm^3; W.B.C., 1,020/mm^3; with a differential count of polymorphonuclear leukocytes, 39%; small mononuclears, 20%; and large mononuclears, 41%.

*Courtesy of J. D. Wheat, Large Animal Clinic, School of Veterinary Medicine, Davis, California.

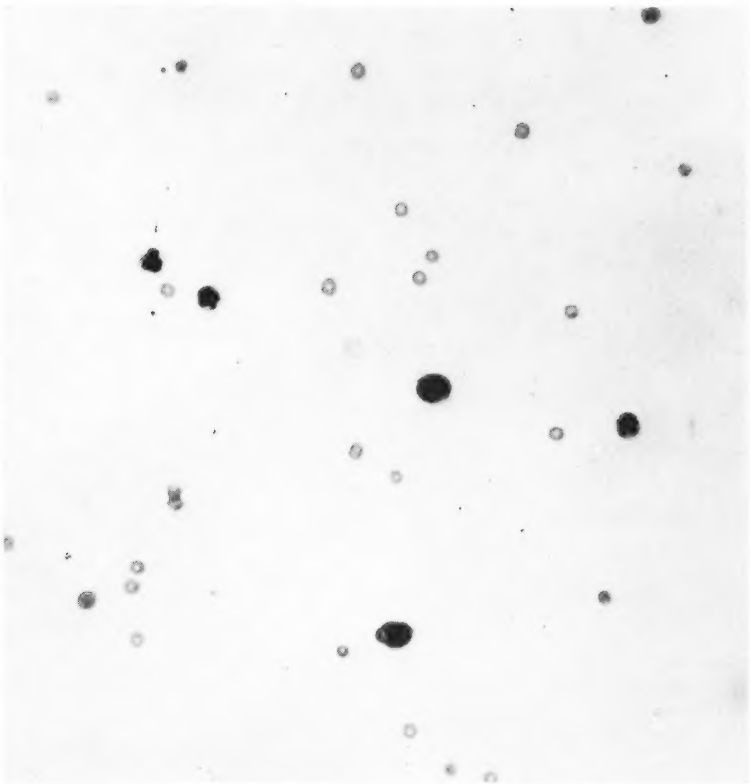
Bibliography

1. Bauer, M. D., Bennett, G. A., Marble, A., and Claflin, D.: Jour. of Exp. Med. **52**, (1930), 835.
2. Bauer, W., Ropes, M. W., and Waine, H.: Phys. Rev. **20**, (1940), 272.
3. Fessler, J. H., Ogston, A. G., and Stanier, J. E.: Biochem, J. **58**, (1954), 656.
4. Bennett, G. A., Bauer, W., and Maddock, S. J.: Am. Jour. Path. **8**, (1932), 499.
5. Davies, D. V.: J. Anat., **79**, (1945), 66.
6. Eggers, H.: Wien. Tierarztliche Monatsschrift, **46**, (1959), 24.
7. Andreyev, P. P.: Veterinariya **25**, (1948), 20.

XUM

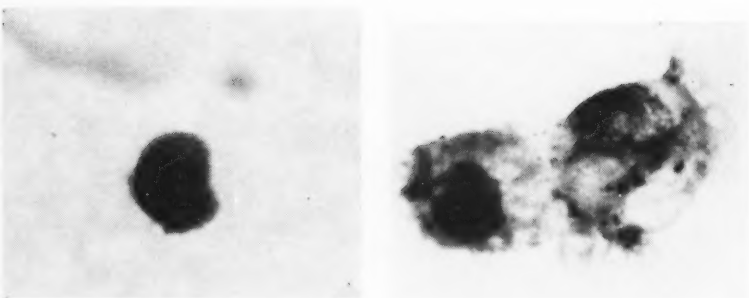


CORNELIUS; Fig. 1. Suppurative carpal synovial fluid of a horse (Group III). Observe the large number of neutrophils present (X400).



CORNELIUS; Fig. 2. Synovial fluid of a horse (Group II). Observe mononuclears and neutrophils present (X400).

XUM



A. B.

CORNELIUS; Fig. 3. Cells from equine synovial fluid (X900). A, normal lymphocyte from a traumatic effusion; B, degenerating mononuclears containing bacteria from a case of spetic arthritis.

CLINICAL BRIEFS

(1) SOME THERAPEUTIC MEASURES

R. COPELAN

(2) "TYING-UP" IN HORSES

J. H. FRIES

DR. COPELAN:

"Cracked Heels" is a condition which is often presented in race-track practice. This condition, localized to the flexor surface of the fetlocks, varies in severity.

The disease may be severe enough to cause lameness in horses in which posterior digital neurectomy has been performed or whose forelegs are affected by a generalized filling or edema from the coronary band to midway up the metacarpal region.

The etiology is unknown but most of our cases respond generously to an application of 125,000 units of streptokinase-streptodornase which has been reconstituted with 5 cc of sterile water and then mixed with carboxymethylcellulose jelly*. This jelly is shaken well and applied to a 4 x 4 gauze sponge which is placed on the affected area and secured by a cotton bandage placed well down over the back of the coronary band so that the jelly may not escape. This is left in place for 24 hours. The results have been quite dramatic with almost total reduction of swelling and pain and a drying effect of the weeping area.

In more severe cases, this procedure may be repeated but instead, an antibiotic cream or ointment is usually applied daily under bandage until recovery.

Another equally promising drug which we have had only limited opportunity to test, is Parke Davis' Elase. This ointment is also an enzymatic debriding agent and is supplied in a one ounce tube. It is therefore handier to use than the preceding drug and if the results prove equal, it will be our treatment of choice.

Several times in the past we have had horses on prolonged antibiotic therapy which have suddenly developed severe diarrhea from what we consider to be a sterile gut. We have tried many drugs but our choice of treatment for this diarrhea is one gallon of warmed, cultured buttermilk given by stomach tube. This treatment may be repeated as often as necessary but usually within twelve to sixteen hours the stool begins to take form.

*Varizyme with carboxymethylcellulose jelly is a product of American Cyanamid Co., Princeton, N. J.

XUM

In other acute diarrheas which do not seem to be bacterial in origin and which have no history of antibiotic therapy, we give concurrently 1-3cc of Metropine† intramuscularly and find we are able to manage this syndrome reasonably well.

We have all, at some time or other, been presented with a filly or mare in training exhibiting signs of constant, or nearly constant, estrus. The typical signs are squealing when touched, leaning on the groom while being rubbed, urinating in the starting gate, lying against the side of the gate stall and wringing her tail and propping in the course of morning exercise. Anatomical deviations of the perineal area, are repaired by episioplasty. Infections of the genito-urinary tract are treated appropriately and a thorough rectal examination of the ovaries and uterus is completed. If after all considerations, the aforementioned signs persist, we resort to hormonal therapy in an effort to allay these signs of estrus which have a direct bearing on the value of these animals as racing prospects. As many of you will agree, treatment is not nearly as simple as diagnosis and prognosis is even more difficult.

In the past, we have used pituitary chorionic gonadotropins, progesterone, and Depoprovera* with mild success.

More recently we have adopted a treatment which percentage-wise promises to be better than those previously mentioned.

This consists of implanting a sterile 100 mg progesterone pellet** subcutaneously in the neck of the animal at the level of the wing of the atlas. Usually the right side of the neck is chosen because the mane falls to this side and covers the scar. Interference from the reins is not a factor in this area.

The technic consists of clipping and preparing for surgery an area two inches square, then infiltrating it with 2 - 4 cc of 2% Xylocaine***. A one half inch transverse incision is made through the skin and fascia. A five and one half inch, curved, Halsted mosquito forceps is used to form a small pocket by separating the fascia from the underlying muscle. The implant is placed in the incision and tucked into the pocket. A simple interrupted stitch is used to close the skin incision. No other medication is necessary. This operation can be repeated in 60 to 100 days as is found necessary by the behavior pattern.

---

†Metropine (Methyl Atropine Nitrate) is a product of R. J. Strasenburgh Co., Rochester, N. Y.

*Depoprovera is a product of The Upjohn Co., Kalamazoo, Michigan.

**This is a product of Organon Laboratorise Ltd., Brettenham House, London, England.

***Xylocaine (Astra), brand of Lidocaine hydrochloride, is obtainable from Jensen-Salsbery Laboratories, Inc., Kansas City, Mo.

The management of edematous conditions has been improved in our practice with the use of the diuretic, Vetamox*.

Occasionally, after we have fired several horses during a cool spell, the weather becomes warmer, bringing with it a great number of flies. Since our usual procedure after firing in the fall is the application of a mercury blister without the use of bandages, we are sometimes faced with unusually severe reactions in one or two of these animals. There is an abnormal amount of swelling, a necrotic odor develops and there is some exudation. In this situation we resort to systemic antibiotics and 2 grams of Vetamox powder in the drinking water daily for two to three days. The odor, unusual swellings and accompanying exudation have disappeared and the horses have recovered uneventfully. Last winter, we successfully employed this drug in the treatment of two 2 year old colts suffering from prolonged septicemia with accompanying pulmonary edema. Fluid and oxygen therapy were supplemented with Vetamox daily for 32 days in one case and 45 days in the other.

We have had local tissue reactions following the use of Vigojex** which has a vehicle that seems to react with moisture in the needle or syringe with which it is administered. Vetamox powder, through its diuretic action has been used with success in helping to alleviate the swelling in these instances.

Castrated colts, restricted in exercise because of concomitant fractures, or which are in the process of being walked after firing, have been treated with Vetamox powder in the drinking water. It has helped to reduce preputial and scrotal edema.

In any shipping fever complex in which considerable lung involvement is anticipated, we employ this same drug. It is readily soluble and acceptable to the horse when administered in the drinking water and it produces a decided diuretic action. Instructions should be given not to discard the unconsumed water but to refill from the level drunk during the 24 hour period.

The equine laryngoscope has been used routinely in our practice to diagnose lesions in the nasal passages and pharyngolaryngeal areas. In most animals the passage of the instrument is easy. Rarely do we employ more restraint than a twitch with another man standing on the same side as the operator, placing a hand over the bridge of the horse's face to keep him from suddenly throwing his head. The laryngoscope is then passed in a manner similar to the stomach tube and no lubricant is required.

*Vetamox (Acetazolamide Sodium) is a product of American Cyanamid Co., Princeton, N. J.

**Vigojex, metabolic stimulant, is a product of Pitman-Moore Co., Indianapolis, Indiana.

XUM

We do, however, use a quaternary ammonium compound in warm water which we pass over the shaft of the instrument to create as nearly the same degree of temperature as exists in the turbinate area.

We have found that horses which violently resist the passage of this instrument are rarely improved in disposition by the administration of tranquilizers or hypnotics. Such animals are anesthetized and cast prior to examination in order to prevent injury.

In the past, we have experienced considerable difficulty with horses which have been blistered after the injection of triamcinoline* into tendon sheaths for the relief of tendonitis. This phenomenon may or may not follow the use of any steroids used for this purpose but our experience is limited to the use of Aristocort*, 25 mg/cc.

The technic of injection includes clipping the affected area of the tendon with a No. 40 Oster surgical clipper blade and appropriate cleansing measures used in any intra-synovial procedure. We then use a 23 gauge ½" needle to infiltrate the area subcutaneously and if possible intra-synovially with Aristocort.

The initial results are quite dramatic with reduction of swelling and pain in the tendon. The physical appearance of the tendon after 24 hours shows almost immediate straightening, with optimum results being achieved over a period of four to five days.

When counter irritation is applied within about a three week period following this procedure we have experienced a number of sloughs in the originally affected area. In two of these cases, treatment of the denuded areas was without effect and periods of six to nine months elapsed before epithelization occurred.

In animals that have had the hair clipped in preparation for tendon sheath injections and have not been subsequently blistered, we have noticed that little or no hair growth occurs in the area, leaving quite a landmark for all to see. In other cases however, it was felt that clipping the area prior to injections was objectionable to the client for one reason or another. These animals were scrubbed as thoroughly as possible and an antiseptic pack was applied for 24 hours prior to injection. These horses experienced no loss of hair over the area and had no lack of growth of new hair.

---

*Aristocort (Triamcinoline, Lederle) is a product of American Cyanamid Co., Princeton, N. J.

DR. FRIES:

Observations on many "tied-up" horses give rise to the following conclusions: The disease is more common in young, growing horses especially fillies and mares. It generally occurs after exercise and is most commonly encountered in the spring. An increased incidence is evident in horses maintained on a low nutritional level and it is most likely to be noticed in horses that have "tied-up" before. Opinion has it that the condition is of greater frequence today than it was ten to twenty years ago. It was also noted that the condition seemed to be one of a generalized spasticity and on occasions involved the muscles of respiration. Regardless of the type of treatment, complete rest brought relief to the affected horse.

As a result of these observations and especially because of the following three factors—the occurrence after exercise, in young horses and in those horses in which the condition had appeared previously—it was considered that this could be a metabolic disease. Because of its marked similarity to tetany in the human, calcium deficiency was suspected as the cause.

Relative to calcium metabolism, it is known that calcium is essential for the coagulation of blood; this might explain why some "tied-up" horses bleed at the nose. Calcium is necessary for normal neuromuscular excitability. Calcium is absorbed into the blood stream from the upper gastro-intestinal tract and its absorption is facilitated by Vitamin D and hydrochloric acid. Excessive phosphorus in the form of phosphates and fat inhibits the absorption of calcium. Calcium in the blood is present in three forms—protein-bound calcium, true solution of calcium (a very small amount of the total blood calcium) and calcium carbonate and phosphate. Calcium carbonate and calcium phosphate comprise well over 50 per cent of the ionizable calcium of the blood. Calcium in the blood increases with hydrogen ion concentration. The blood serum calcium level is lowered by poor absorption from the gastro-intestinal tract, when large amounts are being deposited in bone and when there is a lack of Vitamin D.

With decreased ionizable calcium in the blood, tetany results. Therefore, anything that will reduce serum calcium could bring about tetany. It is conceivable that if a horse were to be deprived of adequate calcium it would be more prone to tetany than horses receiving normal amounts. During periods of stress or exercise, the alterations in metabolism and the concomitant changes in blood pH could reduce the ionizable blood calcium and thus precipitate tetany. It should be kept in mind while discussing ionizable calcium of blood, that the horse could have tetany despite a normal total blood calcium level.

Now let us correlate this calcium hypothesis with the known clinical observations already mentioned.

XUM

1. The condition is common in fillies and mares. It is well known that estrogens are water-retaining hormones. During the estrus cycle, the fluctuation in the secretion of estrogens will cause periodic lowered blood calcium. Leg cramps in pregnant women are common but are rare in males. Women's cramps respond well to ionizable calcium. It is well documented that both estrogens and androgens cause deposition of calcium in bones. In addition the estrogenic hormones are fluid-retaining and hence while causing the deposition of calcium in bone they are also causing dilution of the remaining serum calcium. If then the calcium reserve and blood serum calcium level is "borderline", the females are known to be more susceptible than males, but if a male is severely deficient in calcium he will "tie-up" more seriously than will the female.

2. "Tying-up" is common in the spring of the year. The calcium intake may be normal but low Vitamin D is the causative factor. At this time of year, feeds harvested the previous year are getting low in Vitamin D. The animal has been housed for the winter season and its reserves of Vitamin A and D are low.

3. The disease is more common in horses that have "tied-up" before. As a general rule the same conditions that caused the first "tie-up" still exist and the manifestations just repeat themselves.

4. Horses on a low level of nutrition are more frequently affected. Poor nutrition leads to a lack of sufficient minerals, proteins and vitamins for the needs of a quiescent animal and this factor is multiplied many times for the animal undergoing training or racing.

5. It is much more common than ten to twenty years ago. Now, more than ever before, it is usual to feed alfalfa hay to horses. Alfalfa is high in phosphorus as well as in calcium. This extra phosphorus blocks the calcium level in the blood serum. This might account for its frequency today.

6. The condition generally occurs after exercise. Exercise leads to rapid utilization of calcium. Exercise also causes an increase in the hydrogen ion concentration of the blood. This concentration also causes increased phosphates in the blood with a resulting loss of blood serum calcium. Probably we have all had muscle cramps following exercise and these cramps are caused by an acute lack of calcium.

7. "Tying-up" is more common in young, growing horses. The calcuim requirements of growing animals are about double those of adults. Growing bone is the principal user of this added calcium. If the youngster in question is being trained or raced, this is all the more reason for a "tie-up".

No one of the foregoing may account for any horse "tying-up", but a combination may cause the condition in a genetically susceptible animal.

Working on the hypothesis that by increasing ionizable serum calcium, decreasing serum phosphorus and increasing the body reserves of calcium we could probably prevent "tie-up" in a significant percentage of horses, we made a compound. This compound consisted of calcium lactate powder (to offer a good absorbable form of calcium, free from phosphorus), magnesium hydroxide powder and Vitamin A and D powder. Magnesium hydroxide was used because in the intestinal tract it combines with phosphates to form insoluble magnesium phosphate complexes and thus block the absorption of phosphorus. Vitamin A and D powder was used to facilitate optimum amounts of ionizable calcium. With this simple mixture added to the grain twice daily, 38 horses with known histories of "tying-up" were treated. The response was highly significant. Fifty-eight per cent had complete recovery with no recurrence. Twenty-four per cent showed marked improvement; the results were unknown in eighteen per cent of cases. Of this eighteen per cent it is not known if the animals received the medication in the feed. The dosage used in all these tests was 3 tablespoonfuls in the feed twice daily for 4 days than half the dose for the remainder of the treatment time*.

From the foregoing it was concluded that this simple compound could prevent the recurrence of "tying-up" in a significant number of horses.

---

*EDITOR'S NOTE: The speaker did not state the duration of treatment.

XUM

DIRECTORY OF PROGRAM PARTICIPANTS

C. H. Burger, D.V.M.
Practitioner,
Bakersfield,
California

W. D. Carlson, D.V.M., M.S., Ph.D.,
College of Veterinary Medicine,
Colorado State University,
Fort Collins,
Colorado

R. Copelan, D.V.M.
Practitioner,
Lexington,
Kentucky

C. E. Cornelius, D.V.M., Ph.D.,
Dept. of Clinical Pathology,
School of Veterinary Medicine,
University of California,
Davis,
California

J. S. M. Cosgrove, M.R.C.V.S.,
Practitioner
Riverside Clinic,
Palmerstown,
Co. Dublin,
Eire

S. P. Dey, D.V.M.,
Dept. of Surgery,
School of Veterinary Medicine,
University of Pennsylvania
Philadelphia,
Pennsylvania

E. Dillman, D.V.M.,
Practitioner,
Belmore,
New York

T. E. Dunkin, D.V.M.,
Practitioner,
Oak Park,
Illinois

M. E. Fowler, D.V.M.,
Dept. of Medicine, Surgery &
Clinics,
School of Veterinary Medicine,
University of California,
Davis,
California

J. H. Fries, D.V.M.,
Practitioner,
Durand,
Michigan

J. D. Gadd, D.V.M.,
Practitioner,
Cockeysville,
Maryland

C. E. Hagyard, B.V.Sc.,
Practitioner,
Lexington,
Kentucky

E. W. Jones, Ph.D., M.R.C.V.S.,
Veterinary Clinics,
Oklahoma State University,
Stillwater,
Oklahoma

W. O. Kester, D.V.M.,
Brigadier-General, U.S.A.F.
(Retd.,)
Denver,
Colorado

W. G. Magrane, D.V.M., M.S.,
(Med.),
Practitioner,
Mishawaka,
Indiana

J. E. Martin, V.M.D.,
School of Veterinary Medicine,
University of Pennsylvania,
Philadelphia,
Pennsylvania

C. T. Mason, D.V.M.,
Practitioner,
Little Rock,
Arkansas

F. D. Maurer, D.V.M., Ph.D.
Colonel, U.S.A.V.C.
U.S. Army Medical Research Laboratory,
Fort Knox,
Kentucky

W. R. McGee, D.V.M.,
Practitioner,
Lexington,
Kentucky

F. J. Milne, D.V.M., Dr. Med. Vet., M.R.C.V.S.,
Division of Surgery & Obstetrics,
Ontario Veterinary College,
Guelph,
Ontario,
Canada

M. L. Morris, B.S., D.V.M.,
President, A.V.M.A.,
Denver,
Colorado

J. E. Peters, D.V.M.,
Practitioner,
Pasadena,
California

C. W. Raker, V.M.D.,
Dept. of Surgery,
School of Veterinary Medicine,
University of Pennsylvania,
Philadelphia,
Pennsylvania

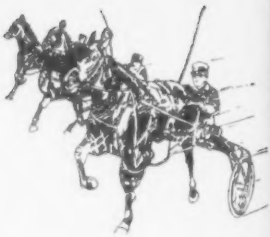
W. O. Reed, D.V.M.,
Practitioner,
Elmont,
New York

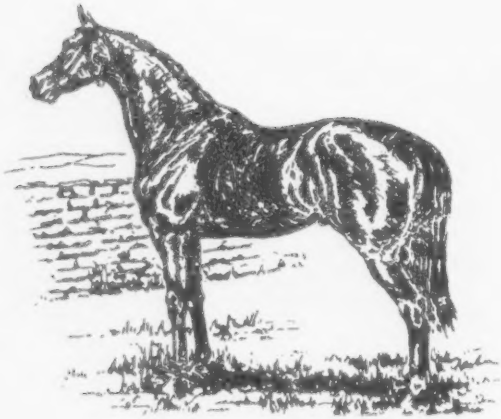
S. F. Scheidy, V.M.D.,
Smith Kline & French Laboratories
Philadelphia,
Pennsylvania

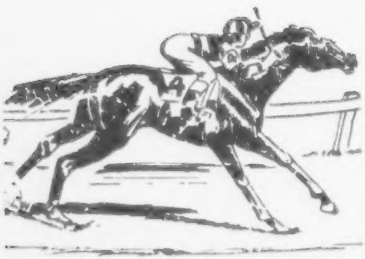
C. Snyder, M.D., F.A.C.S.,
Dept. of Surgery,
University of Miami,
Miami,
Florida

M. B. Teigland, D.V.M.,
Practitioner,
Opa Locka,
Florida

XUM







XUM